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THE ÆTIOLOGY OF ERYTHRŒDEMA (SWIFT'S DISEASE, PINK DISEASE, ERYTHRŒDEMA POLYNEURITIS, JUVENILE ACRODYNIA, TROPHODERMATONEUROSE), WITH SPECIAL REFERENCE TO BLOOD CULTURE.

By W. J. PENFOLD, M.B., Ch.M. (Edinburgh), D.P.H., B.Hyg. (Durham), M.R.C.S. (England), L.R.C.P. (London),

Director of the Baker Institute of Medical Research,

HILDRED M. BUTLER, B.Sc. (Melbourne),
Bacteriologist to the Baker Institute,

AND

I. JEFFREYS WOOD, M.D. (Melbourne).

(From the Children's and Alfred Hospitals, Melbourne, and The Baker Institute of Medical Research.)

PROBABLY the cause of erythrœdema is as yet unknown. Confident assertions have been made by various workers as to its nature; but careful examination of the evidence on which the statements are

based suggests that any dogmatism on the subject is at present premature.

Many authors support the organismal origin of the disease. For example, Vipond,⁽¹⁾ writing in 1922, described a coccus which he had obtained from an enlarged lymph gland in one case of erythrœdema. This coccus grew in the condensation water of human serum medium, the precise nature of which he did not further define. On transplantation from the condensation water to the same medium, a very fine growth of diplococci was observed. The diplococcus was used as a vaccine in the treatment of two patients. The vaccine contained 500,000,000 diplococci to the cubic centimetre, and three doses at short intervals given to each of the two patients are alleged to have caused remarkable improvement, one patient being practically cured and the other very nearly so. In examining the details of the two cases, however, we find a statement to the effect that each patient had been ill for three months before coming under

Vipond's case. Elsewhere he states that both girls were ten months of age at the onset, and fourteen months when they were first seen by him, so that we may say that the patients had been ill from three to four months before they came under his treatment. Before administration of the vaccine, cod liver oil was employed in each case. It was used for two to three weeks in the treatment of the first patient. Its use in neither case was accompanied by improvement. The vaccine was then given, and synchronously with this the remarkable improvement described above set in. Vipond, in his paper, says that the disease lasts four to five months, so that, as far as one can judge from his facts, both patients were treated with vaccine just at a time when they would be likely naturally to recover. On this account, therefore, no great importance can be attached to the results of the vaccine treatment in his cases.

Again, Wood⁽²⁾ (1920) described the finding by Webster of an organism similar to *Bacillus dysenteriae* (Shiga) in a mesenteric gland of a child dead of erythraedema. This organism was not identified serologically, but agreed with the classical organism culturally and biochemically. It was agglutinated by the serum of another patient suffering at the same time from erythraedema. The agglutination was marked in a serum dilution of 1 in 200, and slight in 1 in 500. The second child died from a complicating bronchopneumonia, and no dysenteric or other organisms could be cultivated from its mesenteric glands. From personal communication with Dr. Webster we find that he does not attach any aetiological significance to this organism.

Wood, in the same paper, also records the fact that blood cultures were taken from three cases. Two were sterile after prolonged incubation, and the third yielded a coccus believed to be a contaminant from the skin.

Bacillus dysenteriae (Shiga) infections are not infrequently complicated by peripheral neuritis, so that if it should ultimately be found that infection with *Bacillus dysenteriae* actually occurred in many of these children, some light might be thrown on the causation of the disease.

Rodda⁽³⁾ (1925) reported seventeen cases of acrodynia. His analysis of the effects of treatment are extremely suggestive. In the last six cases of his series excision of tonsils and adenoids was practised as soon as the diagnosis was made, with the result that the average recovery time was three weeks. In a previously treated group of eight cases, in which forced feeding and a generous exhibition of vitamins were undertaken, the average time of recovery was five and a half months. In three of these eight cases tonsillectomy was resorted to, but only after other treatment had failed. The contrast between the results of treatment of these two groups is remarkable and strongly suggests that the disease is essentially a local infection of the tonsils and/or adenoids, in which case the peripheral neuritis present in the disease would be the result of a general toxæmia. If these results are confirmed, a

careful bacteriological examination of these organs would possibly reveal the cause of the disease. Naked eye evidence of tonsillar disease has not usually been discovered, although carefully looked for.

Epidemiological Evidence in Australian Cases.

Shugg⁽⁴⁾ (1931) has described the occurrence of three attacks of erythraedema in two patients from the one family. The first attack occurred in a child of twelve months. After a long illness the patient recovered, to be attacked again, however, at about six years of age. Three months after the commencement of this second attack, a second child, aged about eleven months, fell ill of the disease. This is the only Australian case, so far as we know, in which a second attack arose in one family during the existence of the illness of another member of the family.

From Dr. A. Jeffreys Wood we have received particulars of two other families, each having two cases; but these were separated by long intervals. In the first family there was an interval between the cases of approximately fifteen months, and in the second family one of thirty-one months. If the second cases in these families are due to infection from the first cases, then we must assume that a persistent carrier condition can exist in this disease.

In reference to this question of infectivity, Wood⁽⁵⁾ (1927) mentions a child which developed erythraedema after having been taken by her mother to visit at a home where the baby was already suffering from this same disease. The interval between the visit and the onset of the disease in the second case was one month.

Taking all these four groups together, therefore, we have two where the infection, if it be an infection, may have occurred actually during the course of the disease, and two that cannot have arisen in this way but may possibly have been infected by the earlier patients becoming carriers. Dr. A. Jeffreys Wood, in his communication to us, writes of 128 private patients amongst whom the above four cases occurred. Only twice in all this extensive material has he seen two cases occur in the same family, and, as above mentioned, in both these there were long intervals between the cases.

The available epidemiological data are insufficient to enable any definite conclusions as to infectivity to be deduced. If erythraedema were made notifiable in Melbourne, so that none of the cases escaped notice, it might be possible, from a consideration of the distribution of the cases and the proportion of houses containing pairs of children of the susceptible age group, to determine whether the disease was likely to be infectious.

Chart I represents diagrammatically the facts relative to the above-mentioned seven cases of erythraedema in three families. The length of the lines indicates the duration of the disease in months. Similarly, the spaces between the lines indicate the duration of the intervals between the cases. The number at the commencement of each black line

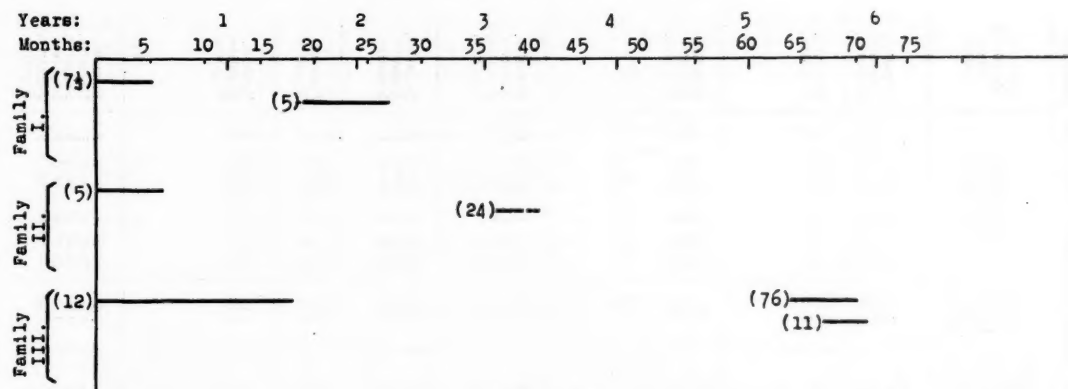


CHART I.

NOTE: The number at the commencement of each black line indicates the age of the child in months at the time of the onset of the disease.

indicates the age of the child in months at the time of the onset of the disease.

Sweet⁽⁶⁾ (1930) described seventeen cases of erythrœdema in which treatment by radiant heat and ultra-violet light was given. His plan was to warm the patient first of all by means of radiant heat, and after that had continued for five minutes, to expose him concurrently to ultra-violet light. In the first treatment the ultra-violet light was applied for three minutes only. The radiant heat was continued for a short indeterminate period after the ultra-violet light had ceased to act. The length of exposure to ultra-violet light was increased in the later treatments. The treatment was given at intervals of three days, and seems to have had a markedly beneficial effect. If Sweet's patients are divided into two groups—those having been ill for two months or any shorter period, and secondly, those having been ill for a longer period than two months—we find that while the average duration of illness in the former group was 3.8 weeks at the commencement of treatment, the number of treatments required before recovery averaged 8.7. In the second group the average duration before the commencement of treatment was 5.6 months, and the average number of treatments required before recovery was 10.7. From this we see, therefore, that Sweet's treatment appeared to be just as effective in the early cases as in the later ones. If the treatment had in reality been without effect, one would not have expected such a result. One would have supposed that the patients receiving treatment late would have recovered earlier, because they were at a stage of the disease when recovery might have been expected naturally.

Other workers have not found the benefit from ultra-violet light that Sweet described as the result of the combined treatment by radiant heat and ultra-violet light; but if Sweet's results be accepted, they do not throw a great deal of light on the ætiology of the disease. Ultra-violet light has been claimed to have a beneficial influence in many infections, and also to have a beneficial influence in certain deficiency diseases.

McCollum and Simmonds (1929), in their monograph on "The Newer Knowledge of Nutrition", state that erythrœdema is not infectious, and give an account of the work of Boas, who, in 1927, published the results of her efforts to produce erythrœdema in rats. The disease was produced by using a full diet, except that the sole source of protein was fresh, crude, dried egg white. After about three weeks of this diet, red scaly patches appeared at the corners of the mouth. The coat became rough, the hair fell out, and red patches of eczematous dermatitis were found on other parts of the body. Frequently blepharitis occurred, so that the eyes were closed. (Edema of the feet sometimes occurred. The rats had a musty smell. Nervous symptoms appeared after six weeks, the limbs became spastic and the back arched. The disease seems to us to be a little different from the erythrœdema of children; for skin hæmorrhages are described as occurring in it, and the redness of the skin in erythrœdema is not of an eczematous character. Moreover, the swelling of the hands and feet is not œdematous in erythrœdema; for pitting cannot be produced by pressure. Further, diminution or loss of reflexes is not uncommon in children, and the pronounced spasticity of the limbs of the rat shows no resemblance to the condition of the limbs in erythrœdema. Moreover, the cause of the disease in rats seems curious. Dried egg white must be the only source of protein, undried egg white not being able to produce the condition. A liberal supply of ordinary egg white to children has not been found to prevent erythrœdema nor yet to cure it.

In the annual report of the South African Institute for Medical Research (1929) reference is made to local work which had been carried out on erythrœdema, which revealed in the blood the presence of *Staphylococcus aureus* and a Gram-positive diplococcus. These organisms were found in the blood in three cases of the disease. The blood was obtained from the fingers by skin puncture and not from a vein. In view of the frequency of infections of the fingers in these cases and the risks of

TABLE I.

Showing the Results of Attempted Blood Cultures in Fifteen Cases of *Erythremia*, with Relevant Data as to the Condition of the Patient and the Stages of the Disease at which the Cultures were Attempted.

Serial Number.	Initials.	Age at Onset.	Duration of Disease from Onset or Commencement of Improvement.	Nature of Onset.	Nature of Pyrexia.	Complications.	Notes.	Date of Taking of Blood.	Approximate Duration of Illness Before Blood was Taken.	Pulse when Blood was Taken.	Temperature when Blood was Taken.	Result of Blood Culture.
1	P.B.	Months. 5	16 weeks. Recovery.	Insidious onset.	No elevation above 37.8° C. (100° F.).	Paronychia.		9/4/31 8/5/31 21/5/31	Weeks. 5.6 5.6 22	132 135 120	38.0° C. (98.4° F.) 37.7° C. (99.9° F.) 36.8° C. (98.2° F.)	Negative. Negative. Negative.
2	B.W.	16	12 weeks. Recovery.	Insidious onset.	Temperature of 38.3° C. (102.0° F.) with complications.	(1) Broncho-pneumonia, otitis media with otorrhoea. (2) Very profuse nasal discharge.		9/4/31 8/5/31	7 11	144 132	38.3° C. (101.0° F.) 36.7° C. (98.0° F.)	Negative. Negative.
3	R.B.	14	32 weeks. Recovery.	Insidious onset.	No pyrexia apart from that of the bronchopneumonia.	(1) Broncho-pneumonia with temperature of 38.9° C. (102° F.). (2) Gross pruritus of body with eczematous rash. (3) Severe scoliosis of lumbar vertebrae. (4) Prolapse of rectum.		1/5/31 8/5/31 12/6/31	23 24 29	132 144 136	38.0° C. (98.4° F.) 37.0° C. (98.6° F.) 36.7° C. (98.0° F.)	Negative. Negative. Negative.
4	V.J.	11	12 weeks. Recovery.	Insidious onset.	Occasional rise to 37.4° C. (100° F.) without complication. Temperature 38.9° C. (102.0° F.) with adenitis.	Suppurative posterior auricular adenitis.		8/5/31 21/5/31 12/6/31	8 10 12 13	132 140 136 156	37.3° C. (99.2° F.) 37.0° C. (98.6° F.) 37.0° C. (98.6° F.) 36.6° C. (102.0° F.)	Negative. Negative. Negative. Negative.
5	J.B.S.	14	18 weeks. Recovery.	Insidious onset.	Occasional rise to 37.5° C. (100.9° F.) without definite complication.	Acute follicular tonsillitis.		10/9/31 20/9/31 29/9/31	12 13 15	100 120 124	36.7° C. (98.0° F.) 36.7° C. (98.0° F.) 37.2° C. (99.0° F.)	Negative. Negative. Negative.
6	A.B.	8	12 weeks. Recovery.	Insidious onset.	Occasional rise to 37.5° C. (100.9° F.).	Nil.	Patient was entirely breast-fed.	11/9/31 16/9/31	6 7	130 132	37.3° C. (99.0° F.) 36.1° C. (100.6° F.)	Positive. Negative.
7	K.K.	8	14 weeks. Death.	Insidious onset.	Four days' terminal pyrexia of about 38.6° C. (102.9° F.).	Four days' vomiting and diarrhoea, followed by convulsions terminating in death.	Post mortem: organs normal except spinal cord (findings of axonal degeneration). During life reflexes present but sluggish. Blood pressure 85/60.	13/9/31 23/9/31 25/9/31 28/9/31 Post mortem.	12 14 14	144 132	37.0° C. (98.6° F.) 40.0° C. (104.0° F.)	Positive. Negative. Negative.
8	E.C.	13	15 weeks. Death.	Onset with attacks of collapse with semi-coma, slow respirations, cyanosis and muscular flaccidity. These attacks, per se, lasted for a week of 10 minutes duration.	Occasional rise to 37.4° C. (99.4° F.) without complications.	(1) Pertussis. (2) Attacks of apnoea and cyanosis apart from coughing. (3) Pericarditis with gross bronchopneumonia.	Tendency towards cyanosis throughout. No cyanotic attacks prior to onset of erythrocytosis. No evidence of structural heart disease. Death from morbilli with frank bronchopneumonia. No necropsy performed.	15/9/31 23/9/31 29/9/31 9/10/31 25/10/31 31/10/31 Post mortem.	8 9 10 12 14 15	128 128 130 132 160	37.2° C. (99.0° F.) 36.7° C. (98.0° F.) 36.9° C. (98.4° F.) 36.7° C. (98.0° F.) 40.0° C. (104.0° F.)	Positive. Negative. Negative. Negative. Negative.

TABLE I—continued.
Showing the Results of Attempted Blood Cultures in Fifteen Cases of Erythraemia, with Relevant Data as to the Condition of the Patient and the Stages of the Disease at which the Cultures were Attempted—continued.

Serial Number.	Initials.	Age at Onset.	Duration of Disease from Onset to Death or Improvement.	Nature of Onset.	Nature of Pyrexia.	Complications.	Notes.	Date of Taking Blood.	Approximate Duration of Illness Blood was Taken.	Pulse when Blood was Taken.	Temperature when Blood was Taken.	Result of Blood Culture.
9	J.B.	Months. 9	12 weeks. Recovery.	Insidious onset.	Rises to 38.3° C. (101.0° F.) without definite complications.	Nil.		25/9/31 8/10/31 23/10/31 19/11/31	Weeks. 8 5 8 11	150 138 100 120	37.3° C. (99.0° F.) 38.1° C. (100.6° F.) 36.7° C. (98.0° F.) 36.9° C. (98.4° F.)	Negative. Negative. Negative. Negative.
10	M.W.	5	12 weeks. Recovery.	Insidious onset.	Nil.	Nil.		23/10/31 28/10/31	12 13	144 136	36.7° C. (98.0° F.) 36.3° C. (97.4° F.)	Negative. Negative.
11	J.S.	8	12 weeks. Recovery.	Acute onset with coryza and bronchitis.	Nil.	Nil.	Slowly recovered after returning home.	4/11/31 11/11/31 29/11/31 9/12/31 11/12/31 18/12/31	4 5 6 8 9 10	128 152 144 136 128 120	37.2° C. (99.0° F.) 37.1° C. (98.8° F.) 36.4° C. (97.5° F.) 36.9° C. (98.4° F.) 36.7° C. (98.0° F.) 36.7° C. (98.0° F.)	Negative. Negative. Negative. Negative. Negative. Negative.
12	N.F.	9	8 weeks. Recovery.	Sudden onset with influenza.	Occasional rises to 37.5° C. (99.6° F.)	Nil.	Patient's condition did not improve but then slowly improved.	9/11/31 11/11/31 26/11/31 20/11/31	4 5 6 7	132 130 128 152	37.3° C. (99.3° F.) 37.3° C. (99.0° F.) 36.8° C. (98.4° F.) 37.2° C. (99.0° F.)	Negative. Negative. Negative. Negative.
13	K.S.	9	10 weeks. Recovery.	Insidious onset.	Nil.	Nil.		13/11/31 24/12/31	5 7	128 136	36.0° C. (96.8° F.) 37.2° C. (99.0° F.)	Negative. Negative.
14	V.D.	16	16 weeks. Death.	Acute onset with "hives" (classical giant urticaria according to mother's history).	Occasional rise to 37.8° C. (100.0° F.) without definite complications. Inter-mittent hyperpyrexia.	(1) Paronychia. (2) Terminal enterocolitis.	Three days before death patient became drowsy and lost consciousness. Oils and dyspnoea developed. Towards the end the stools were liquid and offensive. No vomiting. <i>Post mortem:</i> congested mucous membrane of ileum and proximal colon with enlarged mesenteric glands. Probably an acute enterocolitis. Other organs normal to macroscopical examination.	20/11/31 12/12/31 <i>Post mortem.</i>	13 16	132 —	36.9° C. (98.4° F.)	Negative. Negative.
15	T.G.	5	12 weeks. Recovery.	Insidious onset with slight coryza.	Rise to 37.4° C. (99.4° F.) with enteric upset.	Mild enteritis lasting three days.	Observed in the out-patient department mostly as patient was breast-fed.	23/11/31 2/12/31	6 7	138 140	36.7° C. (98.0° F.) 37.0° C. (98.6° F.)	Negative. Negative.

this technique, it is difficult to assess the value of the results. This Gram-positive diplococcus described was recovered also from the faeces, urine, tonsils and glands. The details of this research have not yet been published, and we anxiously await them.

The Blood Picture in Erythroedema.

It is usually stated that there is a definite leucocytosis in the disease. According to Paterson and Greenfield⁽⁷⁾ (1923), the leucocyte count is from 15,000 to 40,000. In the paper on the subject by Rodda, however, no leucocytic count higher than 20,000 is given as occurring in the cases in which blood counts were made, and the average of his counts from fifteen cases we find to be approximately 12,000.

Again, the nature of the excess leucocytes is variously stated by different authors, some speaking of it as a granular leucocytosis essentially, and some lymphocytic. In view of the fact that at about one year of age the normal child has 15,000 leucocytes per cubic millimetre of blood,⁽⁸⁾ it seems doubtful whether a marked leucocytosis is an essential portion of the clinical picture. Until further blood counts have been carried out, no deductions can be made from them.

A consideration of the salient features of the literature inclined us to favour the organismal cause of the disease. We considered that in investigating the disease from this point of view it would be well to do thoroughly a series of blood cultures. If by any chance the disease should prove to be a septicæmia, then we might obtain cultures without great risk of contamination and relatively easy to investigate. In this paper we give an account of that work.

The cases selected for the investigations were all typical examples of erythroedema. The diagnosis in twelve cases was confirmed by members of the honorary medical staff of the Children's Hospital, Melbourne, and in the three others by honorary medical officers of the Alfred Hospital.

All cases showed irritability, insomnia, anorexia and failure to gain in weight, loss of muscular tone, bouts of sweating and the appearance of a sudaminal rash on the trunk. The photophobia, atonia and general loss of motor power gradually brought about the classical attitudes in erythroedema, namely, the face buried in the pillow, with the eyes taking an occasional surreptitious glance at the neighbours; the hands drooping, scratching the trunk or wandering into the slobbering mouth; the legs sprawling out or tucked under the abdomen. The hands and feet were cold, swollen and red, occasionally verging on blue. The deep reflexes were in most cases present, but sluggish. It was unusual to find them completely absent. The sad, dejected, tolerant expression giving way to storms of fury is a feature of the disease which makes it one of the most distressing encountered in the study of sick children. The patients came from both town and

country, and the diet comprised reasonable ingredients with an adequate supply of vitamins. Two patients, A.B. and T.G., were entirely breast fed (see Table I). Gastro-enteritic disorders were not commonly encountered.

The history of an acute onset was given in some of the cases, but these may have been the result of defective observation on the part of the mother. The patient, E.C., was interesting in that signs of medullary failure were present, but the developing pertussis may have been the cause. The attacks were unassociated, however, with spasms of coughing.

The Technique of Blood Culture.

In most of the cases the blood was obtained from the superior longitudinal sinus. The hair was shaved from the region of the anterior fontanelle and a sterile sheet with a circular hole in it, 6.25 centimetres (two and a half inches) in diameter, was placed in position to avoid contamination from the surrounding hair. The child was rolled in a sheet to restrict any movement of the arms and legs, and the head was firmly held so that the sagittal sinus was at right angles to the floor, the face pointing upwards. The skin was sterilized by the application in sequence of a 1% solution of lysol, absolute alcohol and ether. These materials were dropped on the skin from a drop bottle and gently rubbed in by means of sterile cotton wool swabs manipulated by dissecting forceps. A ten cubic centimetre "Vim" syringe and a "number 12" needle were employed. The swabs, syringe and needles were sterilized in the autoclave after packing them in large glass test tubes fitted with cotton wool plugs, fifteen pounds pressure being used for twenty minutes. When taking the blood, the needle was directed towards the occiput and inserted through the skin in the mid-line at an angle of about 30° to the surface and about six millimetres (a quarter of an inch) anterior to the posterior limit of the fontanelle. The sinus was usually entered at a depth of six millimetres (a quarter of an inch), and ten cubic centimetres of blood were withdrawn. This was immediately injected into the various culture media. Due precautions were taken throughout the procedure to flame the mouths of the containers. In the few cases where the anterior fontanelle was closed, the external jugular vein was employed, the child being held so that the head was at a lower level than the trunk.

The Media Used.

In the first four cases the media used were the following.

1. Hartley's modification of Douglas's tryptic broth.⁽⁹⁾
2. Martin's peptone broth.⁽¹⁰⁾
3. Anaerobic tryptic broth.

This is made from tryptic broth (Hartley) by the addition of a little glucose and cooked meat. It is then sealed with paraffin. One or two days before use active trypsin is added to it. In the interval it is incubated to demonstrate its sterility.

In the remaining eleven cases Hartley's tryptic broth was replaced by Kendall's *K* medium,⁽¹¹⁾ made as follows.

Human brain was extracted with alcohol and benzol, ground to a fine powder, suspended in Tyrode's solution and autoclaved on two successive days. Immediately before use the medium was boiled for five minutes, then rapidly cooled.

Martin's peptone broth and anaerobic tryptic broth were used in 15 cubic centimetre quantities, Hartley's tryptic broth in 100 cubic centimetre quantities, and *K* medium in 30 cubic centimetre quantities.

Three cubic centimetres of blood were added to each medium. All cultures were incubated for two months or longer. Control tubes of uninoculated media were incubated and examined along with the blood cultures so that our media and technique of handling the cultures would be rigidly controlled.

Examination of Blood Cultures.

During the period of observation the cultures were examined daily, and when any cloudiness appeared, smears were made. When cultures became cloudy, but no organisms were detected in the smears, then fresh smears for examination were made three times a week during the first month and twice a week during the second month.

If no cloudiness had appeared by the end of the first three weeks, smears were made and examined. If no organisms were found, examination was repeated at the same intervals as for the cloudy cultures.

Results.

No growths were obtained from the use of Hartley's tryptic broth, Martin's peptone broth and anaerobic tryptic broth. From three out of the eleven cases where *K* medium was used, four positive results were obtained (see Table I).

The condition of the patients at the time of the taking of the blood from which the successful cultures were made was as follows.

From patient VIII the blood for culture was taken in the eighth week of the disease, the patient at this time suffering from pertussis. From the same patient, in the twelfth week of the disease, the pertussis being practically over, another positive blood culture resulted, and apparently on each occasion the same organism was obtained. Patient VI had no complication at the time the blood for culture was taken. Patient VII had an abscess of the leg, but no other complication.

The details of the four positive cultures are set out in Table II.

Growth was said to be detected in the original culture tube only when organisms were observed in smears stained by Gram's method with suitable counter stain. Subcultures on solid media were examined macroscopically daily. In the case of three of the four primary cultures, attempts at subculture made on the first detection of the organisms were unsuccessful. Later subcultures, however, grew slowly, but well.

Subcultures were made aerobically and anaerobically on ordinary agar and on blood agar. Growth was slower in the anaerobic subcultures, and agar appeared to be a more suitable medium than blood agar. The bacteria grew very slowly for the first three or four subcultures, but after six to eight subcultures a good growth on agar was obtained in three to four days. The growth on agar was yellow, sticky and rough, but quite moist. Morphologically the organisms were coccoid, showing marked variation in size and shape, especially when first seen in the *K* medium, in which some of the organisms had the appearance of small diphtheroids (see Figures I, III, V and VII). The organisms were strongly Gram-positive.

None of the four cultures in *K* medium was found to contain more than one type of organism. The pleomorphism characteristic of the growth in *K*

TABLE II.

Positive Blood Cultures, showing their Slow Growth and the Slow Growth of their Subcultures.

Number.	Initials.	Duration of Disease.	Primary Culture on <i>K</i> Medium.		Subculture on Agar Medium.			
			Time from Taking the Blood to the Detection of Growth.	Microscopical Appearances.	Time from Taking of Primary Culture to Making of First Successful Subculture.	Time Taken for Growth to Appear.	Macroscopical Appearance.	Microscopical Appearance.
6	A.B.	6 weeks.	42 days.	Irregular Gram-positive cocci. (See Figure I.)	63 days.	16 days.	Small lemon-yellow opaque moderately sticky colonies.	Irregular Gram-positive cocci. (See Figure II.)
7	K.K.	12 weeks.	14 days.	Irregular Gram-positive cocci, some forms suggestive of a short diphtheroid. (See Figure III.)	21 days.	7 days.	Small lemon-yellow opaque sticky colonies.	Very irregular Gram-positive cocci. (See Figure IV.)
8	E.C.	8 weeks.	35 days.	Irregular Gram-positive cocci. (See Figure V.)	35 days.	12 days.	Small lemon-yellow opaque and very sticky colonies.	Gram-positive cocci. (See Figure VI.)
8	E.C.	12 weeks.	35 days.	Irregular Gram-positive cocci. (See Figure VII.)	49 days.	10 days.	Small lemon-yellow opaque and very sticky colonies.	Gram-positive cocci. (See Figure VIII.)

medium could not be got rid of by repeated plating and the selection of single colonies, but when these organisms were grown on agar they showed a more uniform coccoid morphology (see Figures II, IV, VI and VIII).

Figure IX shows strain "KK" from case VII as it appears in an old culture on agar. There was a good growth in four days, but the photograph was not taken until after twenty-eight days' growth. In consequence, numerous involutionary forms are visible.

In many of the photographs the pleomorphism suggests the commencement of involution in the elements, but whether this is sufficient to account for the appearance of short bacilli resembling diphtheroids we are not yet able to say.

The four strains failed to produce any visible change in milk or to ferment any of the following carbohydrates: isodulcitol, arabinose, xylose, glucose, laevulose, mannose, galactose, maltose, lactose, saccharose, raffinose, inulin, amygdalin, glycerin, erythritol, adonite, dulcitol, mannitol and sorbitol. The sugars were contained in peptone water media. The organism grew in peptone water alone, but took seven days to do so. The growth distinctly increased during the second week. The observation period for the sugars was one month.

Sera were prepared in rabbits from strains "KK" and "EC₁" (see Table I). Three injections, each of one cubic centimetre, of a fourteen day culture in tryptic broth were given intravenously at weekly intervals, and the rabbits were bled one week after the last injection. Agglutination tests with these two sera were carried out against emulsions of the strains "KK", "EC₁", "EC₂" and "AB". Complement fixation tests were also done with these two sera. The antigens were thin emulsions of the four strains, "KK", "EC₁", "EC₂" and "AB". The agglutination results are set out in Table III. These results suggest that strains "EC₁" and "EC₂", taken from the same patient at an interval of about three weeks, were similar, from which we conclude that they did in actual fact come from the blood stream. Although the serum "KK" was weak, the agglutinations of strains "KK" and "AB" were similar, while control normal rabbit serum had a titre of less than one-quarter of that of the specific serum. These

strains, therefore, appear to be related. These agglutination results were confirmed by complement fixation tests.

Strain "AB" was agglutinated by the patient's serum only at a dilution of one in twenty. Three control sera failed to agglutinate the strain at this dilution. Complement fixation with the patient's serum did not occur.

Unfortunately the growth of strains "KK", "EC₁", and "EC₂" was not detected until after the death of the patients, so we have no record of agglutination or complement fixation tests with these patients' sera. The three strains, however, were tested against sera from patients P.B., R.B., A.B., J.B.S. and J.B.; but neither agglutination nor complement fixation occurred.

Preliminary Tests on the Pathogenicity of Strains "KK" and "EC₁" for Young Rabbits and Guinea-Pigs.

The strains were grown in tryptic broth and in K medium for fourteen days. One cubic centimetre quantities were injected intraperitoneally into young guinea-pigs, none of which died or lost any weight.

The cultures were also injected intravenously in two cubic centimetre and one cubic centimetre quantities into young rabbits (four weeks old), three litters of six each being used. The results are shown in Table IV.

Although the numbers are small, it would appear that strain "KK" grown in K medium is probably pathogenic for young rabbits. Grown in tryptic broth, with the quantities employed, it is without effect, at least up to two months. This is of interest in view of Kendall's results with the influenza coccus, which was pathogenic for rabbits only if grown in K medium.⁽¹²⁾

Strain "EC₁", in the quantities used, does not appear to be highly pathogenic, even when grown in K medium.

So far we have not observed any of the characteristic signs or symptoms of erythredema in any of our inoculated animals.

Animal Inoculations with Patient's Blood.

Blood in one cubic centimetre quantities from patients K.K., J.S., A.B. and J.B. was inoculated intraperitoneally into young guinea-pigs, two being

TABLE III.
Agglutination Results with Rabbit Sera "KK" and "EC₁."

Emulsion.	Serum Dilutions.															
	Rabbit "KK."							Rabbit "EC ₁ ."							Control Rabbit.	
	1/20	1/40	1/80	1/160	1/320	1/640	1/1280	1/20	1/40	1/80	1/160	1/320	1/640	1/1280	1/20	1/40
Strain "KK"	+++	+++	+++	-	-	-	-	-	-	-	-	-	-	-	-	-
"EC ₁ "	-	-	-	-	-	-	-	+++	+++	+++	+++	+++	+++	+	-	-
"EC ₂ "	-	-	-	-	-	-	-	+++	+++	+++	+++	+++	+++	-	-	-
"AB"	+++	+++	+++	-	-	-	-	±	-	-	-	-	-	-	-	-

NOTE.—No agglutination occurred when saline solution was used as a control. Incubation was at a temperature of 56° C. for 18 hours.

TABLE IV.
Pathogenicity of Strains "KK" and "EC₁" for Young Rabbits.

Rabbit.	Dose.	Result.	
Litter I.	1 2 c.cms., K medium.	Gained weight steadily.	Weight stationary. Gained weight for three weeks, then lost.
	2 1 c.cm., K medium.	Gained weight steadily.	
	3 2 c.cms. of culture of strain "KK" in K medium.	Animal stolen.	
	4 1 c.cm. of culture of strain "KK" in K medium.	Died 27 days after injection.	
	5 2 c.cms. of culture of strain "EC ₁ " in K medium.	Died 27 days after injection.	
	6 1 c.cm. of culture of strain "EC ₁ " in K medium.	Gained weight steadily.	
Litter II.	7 2 c.cms., K medium.	Gained weight steadily.	Gained weight for four weeks, then lost. Gained weight for three weeks, then lost.
	8 1 c.cm., K medium.	Gained weight steadily.	
	9 2 c.cms. of culture of strain "KK" in K medium.	Died 37 days after injection.	
	10 1 c.cm. of culture of strain "KK" in K medium.	Died 27 days after injection.	
	11 2 c.cms. of culture of strain "EC ₁ " in K medium.	Gained weight steadily.	
Litter III.	12 1 c.cm. of culture of strain "EC ₁ " in K medium.	Gained weight steadily.	
	13 2 c.cms., tryptic broth.	Gained weight steadily.	
	14 1 c.cm., tryptic broth.	Gained weight steadily.	
	15 2 c.cms. of culture of strain "KK" in tryptic broth.	Gained weight steadily.	
	16 1 c.cm. of culture of strain "KK" in tryptic broth.	Gained weight steadily.	
	17 2 c.cms. of culture of strain "EC ₁ " in tryptic broth.	Gained weight steadily.	
	18 1 c.cm. of culture of strain "EC ₁ " in tryptic broth.	Gained weight steadily.	

NOTE.—All injections were given intravenously. Cultures were grown for 14 days at 37° C. The animals were observed for two months.

used for each case. The guinea-pigs suffered only a slight loss of weight similar to that lost by pigs receiving blood from normal children. No inoculation of rabbits was undertaken.

Control Blood Cultures.

Control blood cultures were made from thirty-six children between the ages of four months and eighteen months. The children were usually convalescing after one of the common acute diseases of childhood such, for example, as dysentery or pneumonia. The patients, however, were not an ideal control, as they were not all suffering from a long-standing condition of lowered vitality, such as is usually seen in erythrædema. They were considered, therefore, less likely to harbour secondary invaders of the blood stream.

The technique and culture media were the same as used in the cases of erythrædema. Only twenty-one of the thirty-six had the blood taken into K medium. The subsequent examinations of the cultures were exactly similar to those used in the erythrædema cases. Positive results were obtained in only two cases. A diphtheroid organism grew in one case and *Staphylococcus aureus* in the other. The diphtheroid organism was present in all three culture media, namely, K medium, Martin's peptone broth and anaerobic tryptic broth, and it was detected after two days' incubation. The *Staphylococcus aureus* was isolated from K medium and from anaerobic tryptic broth after fourteen days' incubation. Subcultures of both these organisms on ordinary agar grew readily. They bore no similarity to the extremely slowly growing organisms that we had obtained in the three cases of erythrædema. The diphtheroid organism grew well in subculture in twenty-four hours, and the *Staphylococcus aureus* in forty-eight hours.

The significance of the strains isolated in the cases of erythrædema must be left in doubt. They may be secondary invaders of the blood stream, gaining entrance to it owing to the depressed vitality of the children, or their presence may be actually dependent on the specific disease.

Summary.

Blood cultures have been attempted in fifteen cases of erythrædema. In four of these Martin's medium, Hartley's medium and anaerobic tryptic broth were used in each case, while in the succeeding eleven, Martin's medium, anaerobic tryptic broth and Kendall's K medium were used.

The blood was taken at various stages of the disease, and on several occasions from the individual patients when opportunity offered.

No growth was obtained with any of the media, except with Kendall's K medium, which yielded four positive results in three cases. The blood in the other eight cases yielded no growth.

The characters of the organisms isolated are described.

Organisms in two of the cases appear to be related to one another.

The evidence in hand is insufficient to indicate whether the organisms in question are aetiologicaly related to the disease or not.

Conclusions.

1. We have not found any evidence to suggest that erythrædema is usually a septicæmia.

2. From the blood of three patients organisms have been isolated which are so peculiar as to merit further examination.

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THE CHRONIC DIARRHŒAS: THEIR ÆTIOLOGY AND TREATMENT.¹

By ALLAN S. WALKER, M.D., Ch.M. (Sydney),
Honorary Assistant Physician, Royal Prince
Alfred Hospital, Sydney.

THIS discussion starts where the study of medicine should begin and end, with the patient; for it concerns a purely clinical symptom, chronic diarrhœa.

CLASSIFICATION.

Ideally, classification, like treatment, should be ætiological; but this high ideal can only be partially realized in a subject such as this. However, we must have some working classification, even if it is like many such in medicine, not a true scientific thoroughbred, but a hybrid, in part ætiological and in part symptomatic. Therefore, as a working basis I suggest the following.

I. Diarrhœas Due to Infective States in the Bowel.

- (i) Amœbic dysentery.
- (ii) Bacillary dysentery.
- (iii) Bilharzial dysentery.
- (iv) Certain other protozoan forms.
- (v) Tuberculous enteritis.
- (vi) Syphilis of the bowel.
- (vii) Catarrhal enteritis and colitis.
- (viii) Ulcerative colitis.

II. Diarrhœas Due to Causes other than Infective States in the Alimentary Tract.

A. General:

- (i) Associated with general infective states (for example, septicæmia).
- (ii) Associated with toxic states (for example, exophthalmic goitre, uræmia, Addison's disease).
- (iii) Sprue.

B. Local (that is, due to local causes in the alimentary tract):

- (i) Irritation by food:
 - (a) Due to essentially irritating foodstuffs.
 - (b) Due to secretory defects (for example, gastrogenous and pancreatic diarrhœas).
 - (c) Due to absorptive anomalies (for example, celiac disease).

(ii) Obstruction and/or local irritation.

- (a) By hard fæces (functional constipation).
- (b) By an organic lesion (stricture, polypi, cancer).

(iii) Exaggeration of the nervous excitability of the bowel.

- (a) Nervous and lenteric diarrhœas.
- (b) Spastic colon and muco-membranous colitis.

There are obvious defects in this list. Thus there must be many instances where infective and non-infective causes coexist. Also there may be several factors of totally different type in many individual cases. Certain diarrhœal diseases are hard to place, sprue, for example, in which there are not only disturbances of a general nature, but secretory or absorptive defects as well, not to mention the super-added element of infection still believed in by some authorities.

INVESTIGATION.

Preliminary Considerations.

Now let us consider the individual patient who presents himself with a history of a diarrhœa which is chronic, that is, persisting constantly or intermittently over a period of many weeks, months, or even years. What pointers have we in diagnosis? First, has he diarrhœa? The term is not synonymous with multiple daily defæcation, for this may be a matter of habit, or more frequently the result of incomplete evacuation. If the stools are not loose and if there is no evidence that food or food residue is passed with undue speed through the bowel, the patient has not diarrhœa. If doubt exists, the speed of the intestinal movements may be checked by administering charcoal and observing the time taken to pass through.

Here it is useful to recall that an opaque meal is observed by means of the fluorescent screen to reach the ileo-caecal sphincter in three to four hours after it has begun to leave the stomach. The small intestine, though functionally extremely important in the processes of digestion and absorption, has a huge surface area, and is therefore built for speed. Delay occurs normally from this point onwards, the meal appearing in the caecum about four and a half hours after leaving the stomach, and not arriving in the pelvic colon for eighteen hours. The colon is designed for power rather than speed, and is supplied with that necessary extra lubricant that its heavy loads demand. Although the large bowel is not necessarily the only source of mucus in the stools, it naturally follows that the appearance of excessive mucus indicates colonic irritation.

History.

Now, assuming that the patient has a true chronic diarrhœa, a consideration of the history may help. How long has it lasted? Is it intermittent or continuous? Did it follow an acute attack with a febrile illness? Has it any relation to food, time of day, worry *et cetera*? Does it alternate with constipation? The answers to these questions will not settle the diagnosis, but merely narrow the field.

¹ Read at a meeting of the Section of Medicine of the New South Wales Branch of the British Medical Association, June 16, 1932.

Examination.

Physical examination may reveal points of importance, such as local tenderness, a contracted and readily palpable colon, a tumour, jaundice, secondary anaemia, loss of weight, other systemic disease, or suggestive indications of a neurosis. The next point is probably the Achilles's heel of the clinician, the examination of the stool by simple inspection. I think we must admit that too often we fail to look for ourselves. Such an examination may provide answers to the following questions.

Is the stool frothy, suggesting possibly a carbohydrate dyspepsia?

Is it unusually offensive, suggesting perhaps excessive putrefactive change?

Are there evidences of undigested food? The presence of undigested food is always abnormal, for the bulk of the normal faeces is made up of cellular *débris* and bacteria, and they should contain no unsplit food substance except certain resistant components like cellulose.

Is it fatty, and perhaps unusually bulky and pale, pointing to deficient fat digestion or absorption?

Is it green or unusually light in colour, suggesting a variation or absence of the usual bile-derived pigment?

Lastly, does it contain mucus in excess, or, still more important, blood or pus?

With this information one should now be able to make a surmise at the most likely group of causes concerned; further investigation by radiological, bacteriological, chemical and other methods is pursued when required.

MATERIAL.

I do not propose to examine each of the causes listed above, but now proceed to discuss the more important, illustrating their nature and the diagnostic and other difficulties by brief case histories. Some of these histories are those of private patients, others are taken from the records of the Royal Prince Alfred Hospital. For the purposes of this inquiry I examined 850 histories of patients treated in the hospital during the last twenty years for complaints diagnosed as enteritis, colitis, or at least some form of diarrhoea. In order to limit the subject no attempt was made to consider the diarrhoeal illnesses of children under the age of two years, which are filed separately. All acute cases were rejected, leaving 288 cases of chronic diarrhoea. This number does not include those filed under such headings as "Carcinoma" or other systemic disease.

On the basis of these cases and my own experience, although the latter has been, I fear, all too inadequate, has been built up the description of the diseases discussed, so that an attempt has been made at least to avoid transcribing text book information.

The 288 cases were grouped as follows:

Spastic colon	31
Definite muco-membranous colitis	5
Secretory disturbances	6
Sprue	2
Constipation causing diarrhoea	15
Food disturbances	3
Nervous and constitutional types	12

Catarrhal colitis, with no evidence of ulceration, following known amœbic dysentery ..	6
Catarrhal colitis, with no evidence of ulceration, following known bacillary dysentery ..	3
Catarrhal colitis, with no evidence of ulceration, following known bilharzial dysentery ..	1
Catarrhal colitis, with no evidence of ulceration, of unknown origin	52
Ulcerative colitis	152

Five cases of terminal ulcerative colitis due apparently to, or at least associated with, uræmia, were excluded from the series.

NON-INFECTIVE DIARRHOEAS.**Nervous Diarrhoeas.**

First, let us take the nervous diarrhoeas. These are of two types: the purely constitutional, such as that occurring in association with the stresses of life (for example, in the case of the nervous student before examinations), and the lenteric type. The latter is due to an exaggeration of the normal gastro-colic reflex, whose stimulus is the entry of food into the stomach, and whose response is a mass peristalsis of the colon. Treatment consists in the administration of bromides and belladonna to curb the excessive vagal activity, and suitable dietetic measures, including the avoidance of hurried meals and irritating food, especially food that is too hot or too cold. Dilute hydrochloric acid may be very useful if the condition depends in part also on a secretory defect. But it must not be assumed that all patients with a pronounced nervous factor in their diarrhoea owe their trouble to such nervous cause alone. Practice makes perfect, even in organic reflexes, and an inflammatory lesion in the bowel, by continued goading of the reflex arc, may affect the phenomenon of facilitation of the reflex. The following is an example:

A girl, aged twenty-three years, of intelligent, nervous type, suffered from intermittent diarrhoea of seven years' standing. It was worse during the menstrual periods and was also increased by stone fruit, tomatoes, gravies and soups. The motions occurred chiefly after meals, and she could usually work all day without interruption. Nevertheless, investigation showed that she had a definite ulcerative colitis, with well marked radiological signs.

Spastic Colon and Muco-Membranous Colitis.

Muco-membranous colitis is often called mucous colitis, a name that should be discarded, for it has not real significance; it is not a colitis at all, but a visceral neurosis. The most striking type includes those patients, almost invariably women, who suffer from paroxysmal attacks in which they pass large quantities of mucus in flakes, masses or strings, occasionally accompanied by casts from the mucous membrane of the large bowel. It is this variety that I here include under the name of muco-membranous colitis, for want of a better name.

Such patients are always introspective and usually complain of the characteristic fatigue and common functional dyspepsia of the neurasthenic. In more than half the hospital cases recorded here these symptoms were sufficiently prominent to be specially featured in the records. The patients are often poorly nourished folk, not seldom of gracile type, and, according to Eggleston,⁽¹⁾ hypothyroidic. The caecum is often tender, the colon firm and

sensitive, sometimes feeling like a hard cable in the left iliac region. The epigastrium often heaves with the exaggerated pulsation of the frequently tender abdominal aorta. The arbitrary vagotonic and sympathicotonic types of Hess and Eppinger are recalled here; for there does seem to be some imbalance between the sympathetic and parasympathetic portions of the nervous system. Autonomic stimulation would account for the increased motor activity and the unusual amount of secretion also.

More than half the patients in the present series were in the third and fourth decades of life, and had complained of symptoms over a period of several years. Constipation was specially noted in two-thirds of the number, a figure also recorded by Ryle.⁽²⁾ All patients with this ailment do not have diarrhoea.

Of the 36 patients, 11 were males and 25 females. Many writers give the proportion of females as much higher. I have the impression that it is not so uncommon amongst men, and Ryle's figures (17 males and 33 females) agree with this. It is, however, significant that all the patients suffering from definite attacks of mucous or membranous colic were women. The passing of intestinal sand is recorded in several of these cases; this is a curious, well known feature of the condition. These attacks have been compared, not inaptly, to bronchial asthma, and are included amongst the allergies by some writers. Certainly the stage of the nervous system in these subjects is set ready for such dramatic seizures, and it might well be that some sensitiveness to protein could initiate an attack. Surely, however, the allergic factor, if present, is only one. I have come across one patient who was much benefited by injections of urinary protease, but as this failed to elicit a skin reaction, its therapeutic effect may easily have been psychical.

The ease with which a diagnosis of a surgical lesion may be made in these cases has often been pointed out. Nine of the patients in this series had lost their appendices, and two were subjected to laparotomy, nothing abnormal being found. Two illustrative cases are the following.

A woman, aged twenty-five years, had suffered from constipation for years. During the previous few years there had been intermittent attacks of paroxysmal low abdominal pain, with the passing of large amounts of mucus and shreds, as well as "sand". She complained of breathlessness and precordial pain, but the cardio-vascular system was normal. Appendicectomy had been performed for this pain three years previously, but she stated that she had been worse ever since.

A neurasthenic young woman, aged twenty-three years, had had her appendix removed for low abdominal pain. Two years later adhesions were separated in an endeavour to relieve the unaltered pain, but she still complained of attacks of pain and passed much mucus from the bowel with occasional membranous casts.

Let the surgeon beware of the spastic colon; it is not likely to bring him credit. But let not the physician treat too lightly the patient apparently suffering from a functional disorder.

A young woman, aged twenty-six years, who had definite inanition, complained of passing loose stools with mucus and sand for nine months. Latterly these had been

offensive. Her condition gradually became worse, with the passing of blood, and later she died in hospital. *Post mortem* examination revealed ulceration and scarring of the colon.

It is certain that infection of the mucosa may occur in persons who have previously had a disorder of bowel function only. I feel sure that the transition from the spastic to the catarrhal type of true colitis, and even to the ulcerative, may take place. And why not? Does not the young allergic asthmatic become emphysematous if his attacks persist frequently and even pass into a state of chronic bronchitis with a definitely infected respiratory mucosa? The next case seems to indicate this transition to a true colonic infection.

A man, aged thirty-four years, had suffered for some time from attacks of epigastric pain that always occurred during the day, and never at night. These recurred at diminishing intervals, and spread later to the lower part of the abdomen. Then he began to have diarrhoea with the attacks and passed much mucus with considerable pain. He lost 6.3 kilograms (one stone) in weight. His reflexes were increased; his abdomen was soft and tender. Then he began to pass watery motions with mucus and blood, and was admitted to hospital. Sigmoidoscopic examination revealed a hyperæmic and oedematous mucosa. There was a slight secondary anaemia; the hydrochloric acid was decreased in the stomach, and X ray examination revealed a smooth colon without haustration. The end state was that of ulcerative colitis.

Treatment of the patient suffering from spastic colon must always include due attention to the general health of body and mind. Sedatives and belladonna are very useful, and the constipation will be helped by a bland lubricant such as paraffin or agar agar, and also by a sufficiency of residue in the diet. I doubt, however, if too much roughage in the food is good; it is often overdone. Oil retention enemata are advised by some and are said to do good. Ordinary bowel lavage should be avoided. Undue attention to the local phenomena tends to produce in these self-centred patients a condition of "colon-consciousness", which may be defined as a form of visceral class-consciousness, which, in the microcosm of the body, produces as much disharmony as its social prototype does in the macrocosm of the world.

Diarrhoea Due to Partial Obstruction or Irritation.

The commonest variety of diarrhoea due to partial obstruction or irritation is that which results from constipation or the abuse of purgatives or both. It may be regarded as a non-infective catarrhal colitis. The most frequent causes of constipation have been given as pills and unpunctuality. The same applies here. An interesting example is as follows, illustrating also the costiveness following bacillary dysentery, with its results.

A man, aged thirty-four years, had suffered from bacillary dysentery while on active service. He had been under treatment practically ever since for alternating constipation and diarrhoea and abdominal pain, worse on exertion. Bacteriological investigations yielded no information of diagnostic value. His diarrhoeal attacks were noted to follow the taking of the rather drastic purgatives to which he was addicted. Treatment was undertaken on the lines thus suggested, with relief.

The other variety may be due to stricture, polyp or carcinoma. Stricture may be syphilitic or may

follow the ulceration of ulcerative colitis, or, more rarely, that of tuberculous enteritis. *Polyposis coli* does not give rise *per se* to any really distinctive features, but affecting the descending and pelvic colon, it is responsible for a severe and intractable type of diarrhoea, often with free bleeding and tenesmus. Diagnosis may occasionally be made by digital examination of the rectum, but more often by the use of the sigmoidoscope. The opaque enema may suggest the lesion also. It may be a very protracted affection. One patient had symptoms for forty years and died of ulcerative colitis. It has a definite association with cancer, as is shown by the figures of W. Susman,⁽³⁾ who recently found polyps in the colon in 6% of a routine series of 1,100 autopsies. In half of all the cases of cancer, polypi were found also, and out of a total of 66 cases of polypi, cancer occurred in 15. Polyps are also found in the bowel in ulcerative colitis at times; there were eight cases recorded in the Royal Prince Alfred Hospital series.

Carcinoma is thought of by every medical practitioner when confronted by a patient at or after middle life complaining of alternate constipation and diarrhoea. But carcinoma does occur without constipation, as the following tragic case shows.

A man, aged forty-two years, had had diarrhoea for a year. The attacks lasted one or two days, and during the previous few months had recurred weekly. There were seven or eight watery grey stools a day, occurring more frequently after meals, but not containing mucus or blood. There was some indigestion. Four years previously he had had similar trouble, and X ray examination had then revealed only visceroptosis. But now he had a mass in the hypochondrium, and an opaque enema demonstrated almost complete obstruction of the sigmoid colon, with pronounced irregularity of the bowel. Laparotomy confirmed the diagnosis of an inoperable cancer of the colon.

Even simple mechanical factors may be of importance, and should be thought of, as the next case shows.

A woman, aged fifty-nine years, had suffered from diarrhoea, with the passage of mucus and, occasionally, blood. Sigmoidoscopic examination revealed no lesion in the mucosa of the bowel. She was unable to retain a barium enema, a fact simply explained by a gynaecologist who saw her in consultation and who later repaired a complete tear through the prineal body. The operation gave the patient great relief.

Diarrhoea Due to Anomalies of Digestion.

Unsuitable food alone may cause diarrhoea. Some diabetics, apart from other causes, may have this symptom owing to the imperfect absorption of a diet rich in fat. Here, of course, the possibility of an additional secretory factor should be recognized.

Among the known secretory defects are those of the stomach and pancreas. Gastrogenous diarrhoea has been the subject of a considerable literature. As a pure entity it is probably rare, being more frequently associated with additional pancreatic defects. I have long sought to cure a patient of his diarrhoea by the simple magic of giving him dilute hydrochloric acid, but notwithstanding the optimism of some writers, I must humbly admit that, though I have seen improvement, a really dramatic success has not so far come my way. One

patient in this series who was reported as being achlorhydric, was apparently much benefited by acid, though the dose given was only one cubic centimetre (fifteen minims) three times daily.

Carbohydrates may fail to digest. There then arises a condition of troublesome flatulence in the intestine, with the passing of frothy, frequent stools. A corresponding disability in the splitting of protein or in the absorption of its fractions may cause the dejecta to be very offensive. These may be in part at least infective disorders and yield to appropriate dieting.

The anomalies of absorption are of great interest. An instructive instance is the following, a case of coeliac disease.

A child, aged eighteen months, addicted to the reprehensible habit of dirt eating, had had for the previous six months a swollen abdomen, suffered from attacks of vomiting and occasional diarrhoea, and latterly, constipation. He was a pale, rickety child; the stool was bubbling, light grey and soapy. There was no radiological lesion in the digestive tract. The dried faeces contained 37% of fat, of which 69% was fatty acid. Thanks to Dr. Edgar Stephen, who undertook the care of the patient in the Royal Alexandra Hospital for Children, he improved on a low fat diet with adequate vitamins.

Sprue requires mention, for it occurs in Australia, chiefly in the more tropical parts, and is apt to be forgotten. As well as a primary malady, it is seen sometimes as a sequel of amœbic dysentery, as in the case recently reported by Crawford and Gutteridge from Brisbane.⁽⁴⁾ The abdominal distension, decreased liver dulness, and the pale, bulky, frothy stools, together with the sore tongue and anaemia suggesting the pernicious type, form a distinctive picture.

INFECTIVE DIARRHOEAS.

Now let us turn to the infective group, first disposing of some of the less common.

Tuberculous Enteritis.

Tuberculous enteritis is not very uncommon in association with the more advanced degrees of phthisis. The relative frequency of ischio-rectal abscess in tuberculous people is a reminder of this. As a terminal lesion tuberculous enteritis is probably quite common, though, curiously enough, it does not always cause diarrhoea; in this respect it recalls typhoid. In this part of the world, where mesenteric and other forms of abdominal tuberculosis are not common, it is not surprising that tuberculosis of the bowel is rarely seen apart from pulmonary disease. Only one case of tuberculous enteritis was specially noted among 744 patients with pulmonary tuberculosis attending the Anti-Tuberculosis Dispensary at the Royal Prince Alfred Hospital during last year. Of course, it need hardly be pointed out that diarrhoea occurring in a subject of phthisis is by no means necessarily due to the same cause as the lung lesion. An instance of the difficulty of determining the reason for a chronic diarrhoea is now given.

A man, aged thirty years, who had served with the forces in Egypt, suffered from troublesome diarrhoea. He had chronic tuberculous disease of the lungs, the clinical

and radiological features of which did not suggest activity and which was not considered to be due to war service. He had passed blood, but none was found in the stool while he was under observation. Repeated examination of the stool revealed nothing of ætiological importance, and it was finally decided that the cause was neither tuberculous nor bacillary infection of the bowel.

Syphilis.

Syphilis is not a common cause of intestinal lesions. The most usually found are the jejunal or ileal multiple gummata and the larger rectal gummata, the latter being a well known cause of stenosis. This problem more often confronts the surgeon than the physician; but diarrhoea sometimes occurs during the treatment of syphilis and may give rise to unrest in the mind of the doctor, as in the bowel of the patient. The following case is one where again the ætiological factors were difficult to assess.

A man, aged twenty-nine years, had suffered from diarrhoea of slow onset on and off for four years. He had never been constive. He was found to have a stricture of the urethra, and his blood serum responded definitely to serological tests for syphilis. After nine months' specific treatment the serum did not react to such tests, but the diarrhoea was worse. No arsenic had been given for some time before. There was a definite nervous factor in his condition. Radiological examination was suggested, but dietetic and medicinal treatment cleared the trouble up before this was done. As he has remained well, it seems as if a specific cause can be excluded.

The Dysenteries.

The tropical dysenteries, with the possible exception of the bacillary variety, are not likely to be of importance in the southern and eastern States of the Commonwealth.

Bilharzial dysentery is not endemic in Australia.

The flagellate protozoans are found, but they are very doubtful offenders. *Trichomonas hominis* and lamblia were each reported as being found on one occasion among 181 patients whose stool was microscopically examined in the present series. Dr. Heydon, of the School of Public Health and Tropical Medicine in Sydney, informs me that lamblia is almost universal amongst healthy institution children examined in Townsville. *Trichomonas vaginalis* is recognized as a cause of vaginal discharge, and has been referred to in recent local literature; but it is very doubtful if the flagellates are pathogenic in the bowel.

Amœbic dysentery is of greater importance. Outbreaks of local nature have occurred, as in Western Australia, and a recent endemic case was reported from Queensland.⁽⁵⁾ But in the temperate portions of the continent at least it is usually imported from other endemic areas. It is interesting that 10% of the population of Britain harbours an amœba indistinguishable from the *Entamœba histolytica*, and Dr. Heydon tells me that he found by repeated stool examination of the children in Townsville orphanage that 50% carried this protozoon, although none of them had diarrhoea. It would seem possible that this may be a similar but non-pathogenic strain, with no bearing on amœbic dysentery; yet this cannot be assumed with certainty. Therefore, although the amœbic disease can probably be set aside as an

important ætiological factor in chronic diarrhoea in this part of Australia at any rate, it should not be forgotten, for tropical diseases may yet give rise to more trouble in this land, especially as transport improves.

Colitis following a known attack of amœbic dysentery, of course, is not necessarily amœbic. The amœbæ and cysts may be found during a period of activity, but when doubt exists, sigmoidoscopic examination will help, as the picture is characteristic.

Bacillary dysentery is a more pressing problem. We know that members of the dysentery group cause diarrhoeas of varying severity in this country, but what part they may play in causing chronic inflammations is obscure. Castellani⁽⁶⁾ ⁽⁷⁾ has described a chronic type of diarrhoea due to what he calls "meta-dysenteric" bacilli. The patients suffer from vague abdominal symptoms and recurring diarrhoeal attacks for long periods, and lose weight. Their blood serum agglutinates the bacilli recovered from the stools. He claims success with dietary and vaccine treatment, but admits that the infection may remain latent.

This subject is really wrapped up in the consideration of ulcerative colitis, and will be returned to later.

We are now left with two classes of inflammatory colitis, catarrhal and ulcerative.

Catarrhal Colitis.

The catarrhal cases numbered fifty-two in this series. They are an ill-defined group, and in this particular classification I have placed a number of cases because, though of infective nature, there was no evidence that they belonged to any other category. Possibly more complete records or investigation might have resulted in a more definite pigeon-holing of these.

Tidy⁽⁸⁾ believes that catarrhal and ulcerative colitis are really indistinguishable, and the only difference is one of degree. It is hard to agree with this view completely, for there are cases in which chronic colitis exists with no demonstrable lesion, and while they appear to be of infective origin, they may yield well to treatment and not evince any tendency to become ulcerative in type. Some of the chronic diarrhoeas of young children belong to this group, but are not specially dealt with here. Some writers speak of "catarrhal ulcers"; this appears to be a contradiction in terms. Among the 52 cases here collected, sigmoidoscopic examination was carried out in 14 and no lesion of the mucosa was disclosed. X ray examination revealed no lesion in 14, diverticulitis in two, spasm in one, a rather smooth colon in one, and stasis and ptosis in one. Many of the patients suffered from recurring looseness of the bowel over a considerable period, but as a rule had general or digestive symptoms of slight degree only. As the management of these cases is virtually the same as that of the lesser grades of ulcerative colitis, treatment need not be dealt with here specifically.

Ulcerative Colitis.

Ulcerative colitis provides the most important and interesting section of the subject.

The following analysis sets forth the outstanding features of the 152 cases collected in this series. As these have been collected from the records of twenty years, it will be realized that the data are often inadequate, especially as regards special methods of investigation in the early years. The value of bacteriological and radiological examinations cannot be estimated from these figures. It is noticeable that the percentage of failures to find lesions by X ray examination diminishes as we reach recent years, owing to better equipment, greater skill and fuller knowledge. The poor pathologist has not been well treated. Most of the stool examinations recorded were made from specimens in as fresh a state as an ordinary routine method of collection would allow, the portions selected being no doubt as a rule selected by a nurse, while direct examination of material taken from a visualized ulcer has only been available on comparatively few occasions, and that only in recent times.

There were 77 males and 75 females in this series. These belonged to the successive decades of life as follows: First, 5; second, 10; third, 52; fourth, 53; fifth, 21; sixth and over, 11. The duration of symptoms was over three years in 25% of the cases and under one year in 40%.

One hundred and eighty-one patients were investigated by microscopical and bacteriological examination of the stools, usually on more than one occasion. Lactose fermenting organisms only were found in 168, streptococci in five, a non-hæmolytic diplococcus somewhat resembling that described by Bagen in two, *Bacillus fecalis alkaligenes* in four, bacillus of Morgan "number one" in six, and flagellates in two.

Radiological examination revealed no abnormality in 21 cases, early changes suggesting a chronic colitis in 13, and well marked changes in 23.

Sigmoidoscopic examination was made of 83 patients. No abnormality was detected in 10; congestion and oedema, often with oozing points, in 40; ulceration in 22; polypi in 8; and stricture in 3.

Description.

The onset of this serious malady is usually insidious. In 29 of the cases an acute onset was definitely noted; in the others the onset, when mentioned, was described as gradual. In a number of female patients the beginning of symptoms was given as related to pregnancy or parturition.

Mucus and blood eventually appear in the stools in variable amounts. The process begins as a rule in the rectum and pelvic colon, and gradually invades the upper parts of the large bowel. The blood is therefore bright and may be quite profuse. One patient lost amounts sufficiently large to contribute to her death. Pus may be in microscopical quantities only, or be observed to flow away between definite evacuations. Pain is present chiefly during

defaecation, and may be severe. There is always some secondary anæmia in chronic cases. Irregular fever is common, and leucocytosis of moderate degree is found in the severer cases during exacerbations.

The nature of the ulceration is the same as in bacillary dysentery. Bagen⁽⁹⁾ describes four phases in the appearance of the mucosa as seen through a proctoscope or sigmoidoscope, namely: hyperæmia, oedema, miliary abscesses, and miliary ulcers. He also notes the presence of scarring and pitting. These changes correspond well with the various pictures described by the different observers in this series. In the early stages the ulcers may appear as cracks or erosions, and later form shallow yellowish areas, smooth or sloughing. It should be emphasized that a diagnosis of ulcerative colitis does not imply the presence of large or even definite ulcers in the bowel. The condition is ulcerative, not of necessity ulcerated at all stages; the ulcer is the end lesion. A point may be noted here: the inveterate nature of the ulceration seen in many cases. The ulcers often tend to heal, but break out again in the same or different places. Scarring is common, tags may remain, and stenosis of the bowel may occur. This tendency to heal and recur is curiously reminiscent of the recurrent tendency of both oral and peptic ulcers, commented on recently at a meeting of this Branch. Either some inherent tendency exists in the tissues or there is some uneradicated infective factor. The inveterate nature of this disease is illustrated by the following brief case histories.

A woman, aged thirty years, with history of ulcerative colitis of six years' duration, was given prolonged treatment, including dysenteric antiserum and kaolin by mouth. She remained well for eighteen months, but then returned with identical symptoms.

A man, aged thirty-two years, previously treated with great relief, returned within a year with a recurrence: the bowel was seen once more to be acutely congested and bleeding at numerous points. This condition responded well to a course of silver nitrate lavage.

A woman, aged twenty-seven years, had suffered from colitis intermittently for seven years. Five years previously an appendicostomy had been performed. On this occasion ulcers were seen in the rectum and were cauterized. She was relieved, but later returned, passing blood and mucus again. Ulcers were again seen, and also a polyp. The former were treated by cauterization; the latter was removed. She returned again *in statu quo*. This time a colectomy was performed and she left hospital well. Later, she once more returned, passing twelve or more bloody stools a day, and had an acute febrile exacerbation lasting two months. X ray examination revealed no lesion, and by use of the fractional test meal she was shown to have a rather low stomach acidity. She recovered, only to return again in a like state.

Surely this would depress the patient, physician and surgeon alike!

The resemblance of the lesions to those of bacillary dysentery has been noted. A further resemblance is the occasional occurrence of the complication of arthritis.

A man, aged twenty-two years, had suffered from profuse mucous and bloody diarrhoea at intervals for two years. The bowel mucosa was seen to be ulcerated and bleeding in the rectum, and X ray examination revealed a lack of haustration. Appendicostomy, performed six months

before, and thorough through-and-through lavage, had improved him very much, but during a local epidemic of acute diarrhoea he had a relapse. While in hospital one knee became swollen, with some effusion and palpable synovia. Three series of injections of dysenteric antiserum were given, each of 90 cubic centimetres, divided over three successive days. One month later pain and swelling occurred in one hip, and later one shoulder. The intervals of time elapsing after the injections of serum were too long for a serum sickness. The stomach contents were of normal acidity, and bacteriological examinations revealed only lactose-fermenting organisms in the stool. He had less than three million red blood cells to the cubic millimetre. After a blood transfusion and seven months of irrigation and general treatment he recovered.

Causation.

Such is the picture of this serious disease that may end fatally in a few weeks or months, or may drag on for years. What is its cause? Frankly, we cannot say with any certainty that we know. Hurst⁽¹⁰⁾⁽¹¹⁾ maintains that it is really chronic bacillary dysentery, and in favour of this urges that even in known chronic cases of this infection it is very difficult to isolate the bacillus. He further quotes a few cases in which the dysentery bacillus has been successfully demonstrated, especially one in which he grew it from the base of a snared polyp. He also advances the successes he claims from the use of dysenteric antiserum. Agglutination tests do not support this hypothesis. Moreover, though improvement does follow the use of a polyvalent dysentery antiserum, it should be remembered that the Shiga type of bacillus is the only member of the group that produces a true exotoxin. This narrows the claim for a true specificity. Moreover, Borgen⁽¹²⁾⁽¹³⁾ claims dramatic results from his allegedly specific diplococcal serum, while Kalk⁽¹⁴⁾ reports good results in the treatment of *colitis gravis* with serum administered so as to cause deliberate anaphylactic symptoms, reinforced by blood transfusions; he sees a direct connexion between the severity of the disturbances due to the serum and the degree of improvement shown by the patients. It is noteworthy that Hurst remarks that the reactions after the administration of serum are often severe. May not the effect be that of protein shock?

Borgen⁽¹⁵⁾ has written a number of articles claiming specificity for his Gram-positive non-haemolytic streptococcus. He adopts the logical procedure of taking cultures from the actual scraped surface of the ulcer, and describes a special technique. He has also produced ulcerating lesions in the bowels of animals by inoculating this coccus. There is, however, nothing even in this evidence to prove that the organism is the real cause of the disease in man and not merely a secondary invader. It has been pointed out that a similar organism has been found on a couple of occasions in the present series.

The importance of the streptococcus as a pathogen is, of course, undoubted, but perhaps, when this energetic member of the bacterial underworld is discovered by the bacteriological police in the neighbourhood of a morbid anatomical crime, suspicion of its guilt may possibly (and perhaps pardonably) be sometimes too readily entertained.

Certain haemolytic members of the *Bacillus coli* group have been incriminated also; but the evidence is very slender. The late lactose-fermenting bacilli, such as those of the Sonne group, are also causing the bacteriologist some perturbation, and it is possible that the common report, "all lactose fermenters", may not have been as innocuous as it has seemed in the past. Avitaminosis and a constitutionally defective bowel have also been suggested as contributing causes.

There is ample room for further work on the subject; let us hope that, with the facilities we hope to have in the future in Sydney, some local accurate work may be undertaken.

Special Methods of Investigation.

This brings us to consider the question of the relative value of the various special methods of investigation and their degree of necessity.

Certain proof of the existence of ulcerative colitis may be gained only by the use of the sigmoidoscope. But is this necessary? The symptoms are distinctive as a rule, if the other forms of diarrhoea are excluded. It is, of course, necessary to exclude haemorrhoids as a source of bleeding and fissure as a cause of tenesmus. Since ulcerative colitis is really a symptomatic diagnosis and not truly aetiological, visual examination of the bowel, though useful, is not indispensable. Polyposis also can only be definitely diagnosed by this means, but even then the problem of treatment is no nearer solution, except that a warning of a possible cancer is given. Examination by X rays after an opaque enema in the chronic cases is very valuable. A study of the case histories of recent years shows that radiology very seldom failed to give information when the sigmoidoscope revealed a lesion of definite severity. The findings are as a rule rigidity, shortening, and often narrowing of the colon with loss of the normal haustration, and hyperirritability. Signs of destruction of the mucosa may be seen, such as irregular flecking of the barium in the ulcerated zones, and polypi may produce a honeycombed effect.

If it is known that in a given case colitis is due to bacteria of some sort or another, it must be admitted that bacteriological examination does not afford much help in treatment. Treatment may be successfully carried out on purely symptomatic lines without such aids. In fact, unless the bacteriologist is given the opportunity of examining a really fresh specimen, truly representative of all parts of the stool, especially those containing mucus and blood, such examination is a waste of time, energy and money. But it is to the bacteriologist that we look for the elucidation of this problem, and we must see to it that he has adequate opportunities.

Treatment.

Finally there comes the practical side of treatment. In the absence of positive knowledge as to the cause, no really specific treatment is agreed upon in all quarters. But the trial of either the bacterial serum or the diplococcal may be made.

As regards vaccines, Horgan⁽¹⁶⁾ and others have recently claimed cures from the use of a diplococcal vaccine, showing an optimism that does not seem to be shared by other observers. I am decidedly sceptical as to the value of any bacterial vaccine in such conditions, since the present place of all vaccines in the field of therapeutics is so very precarious.

The diet in general should be of low residue type. Roughage should be absolutely forbidden, especially during the acute period. The calories and vitamins of the food should be abundant. Acidophilus milk may be a helpful addition to the diet. Rest in bed and warmth are needed in the acuter and severer types. But, as Barga points out, when the patient begins to improve, he should be encouraged to get up, as a physical tonic and as mental encouragement to escape from the "toilet-stricken" outlook.

The most sane and valuable line of general treatment laid down in recent literature is that of Tidy.⁽⁸⁾ The particular value of his plan is that it can be carried out by any medical practitioner with reasonably good nursing facilities, and does not need any elaborate diagnostic or therapeutic aids, however ideal these may be. The diet advised is one containing ample fluids, to offset the inevitable losses from the body. Light puddings, eggs, fish, butter in moderation, meat jellies and extracts, cereals, fruit juices, and not too much milk, are given.

The local treatment comprises enemata of three varieties: purely sedative (starch and opium), bland (salt solution and bicarbonate of soda), and medicated (containing a silver salt). The principle is that the starch and opium enema is used until comparative calm reigns in the colon. Then bland irrigations are tried and continued until the stools number less than five in the twenty-four hours. If acuter symptoms or intolerance arise, the sedative stage is returned to as many times as may be needed. Great care is taken that the anus and rectum do not become sore, by not overdoing treatment and never giving even an opium enema on more than three successive days. When the patient tolerates the bland washing well (and this tolerance may take months to accomplish in bad cases), the medicated lavage is begun, never being used more often than on alternate days, and retained for short periods at first, gradually increasing to fifteen or twenty minutes. Tidy advises "Albargin", a silver gelatose, 0.9 gramme to 1.5 grammes in 700 cubic centimetres (15 to 25 grains in 25 fluid ounces) of water. It is said to be more effective than silver nitrate, since its action is unimpaired by the presence of organic material. The bowel, of course, must be washed out before the medicated enema is used, and an adequate time allowed for the patient to settle down between the two. This careful *régime* is based on logical ideas, and represents a definite advance on the haphazard scheme of ordering indiscriminate lavage.

Tannic acid solution (1 in 500) and silver nitrate solution (1 in 4,000), increased gradually in strength to 1 in 400, are both suitable for irrigation,

but the patient's tolerance must be carefully studied. Bismuth in large doses is useful, and chalk given as the *mistura cretæ* of the British Pharmacopœia, and kaolin and charcoal, by their adsorptive action, are also valuable. Apart from dilute hydrochloric acid (and about 25% of these patients are stated to be achlorhydric) the aromatic sulphuric acid may be given, and is a useful drug. Blood transfusion definitely helps the severely intoxicated patients.

The last point to be decided in treatment, and a very important one, is the place of surgical measures. Of the 152 patients here studied, 20 were operated upon. The operations performed were: Laparotomy (two), laparotomy with appendicectomy (one), appendicostomy (eight), caecostomy (seven), colostomy (one), and colectomy (one). The results were variable. Some patients improved after through-and-through lavage. But the way is not all plain sailing. In one case the fistula closed as soon as the catheter was taken out. In another troublesome escape of faeces subsequently necessitated the closure of the opening. That colitis may relapse in spite of the facilities thus given for irrigation is shown by several cases in this series. In one case relief was obtained after the performance of appendicostomy, but later the patient relapsed, and the bowel was seen on examination to be once more acutely inflamed, with small areas of the mucous membrane breaking down into ulcers.

Mummery⁽¹¹⁾ believes that appendicostomy is the method of choice and that it is curative if done early. The occurrence of incontinence is regarded as an absolute indication for some form of surgical intervention. There are probably few physicians who would agree with the suggestion that appendicostomy should be done early; but the best argument put forward is the economic, that patients are thereby enabled themselves to carry out irrigation that would dotherwise need a prolonged stay in hospital. However, if irrigation can be carried out in the ordinary way, it is just as effective, and the providing of a surgical stoma is no guarantee against recurrence. As in most of the problems of medicine, each patient must be treated according to the individual indications.

In conclusion I would point out that ulcerative colitis is not a rare curiosity. It should be diagnosed before it reaches the stage of grave organic damage and toxæmia, and patient and sufficiently (but not unduly) energetic treatment undertaken, repeated at intervals, if demanded by actual or threatened recurrence.

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Reports of Cases.

TREATMENT OF A DIFFICULT COLLES'S FRACTURE BY SKELETAL TRACTION.

By THOMAS KING, M.D. (Melbourne), F.R.C.S. (England),
Clinical Assistant, Saint Vincent's Hospital and the
Children's Hospital, Melbourne.

SKELETAL traction (or the powerful and direct pull on bone by transfixion with tongs, pin, or stretched wire), popularized in the first instance with the ice-tong calliper, used for fractures of the femur during the war, has been extensively used on the Continent for a number of years. Strongly advocated by Hey Groves in England for over ten years, it has not been widely adopted there until recently. Though used in the treatment of fractures of the femur and shafts of the leg bones, occasionally the humerus, its wide applicability in the reduction of many types of difficult fractures, particularly when associated with dislocations, of practically every bone of the extremities, including the small bones of the hand and foot, is a recent advance, mainly due to Lorenz Böhler at The Accident Hospital of the Austrian Insurance Companies, at Vienna. Böhler, with an unsurpassed experience of many thousands of fractures, is a strong advocate of non-operative methods of reduction. He condemns as disastrous open reduction, which he believes has sacrificed thousands of lives and crippled far more, though all authorities will not be in agreement with this.

The case described here illustrates the absolute control obtained by skeletal traction. In this case Böhler's method and apparatus for dealing with difficult fractures of both bones of the forearm was applied to a Colles's fracture of five days' standing. Like all other fractures, Colles's type may now be caused by a much greater force, for example, motor car accidents, than was formerly the case. As a result of this greater violence, the type of fracture may be more serious and troublesome to treat, due to comminution and greater displacement of fragments.

This fracture remained unreduced despite several attempts by manipulation and manual traction, and then the following method, obviously only suitable in exceptional circumstances, as in this case, was carried out.

Twenty cubic centimetres of a 2% "Novocain" solution are injected between the bone ends into the "traumatic sac" (Laming Evans) or "hæmatoma" (Braun). Two

cubic centimetres are injected also around the avulsed styloid process of the ulna and the triangular cartilage. Then one cubic centimetre of "Novocain" solution is injected on either side of the olecranon, which is then skewered by a rustless pin or by Kirschner's chromium plated piano wire. The site of transfixion is two fingers' breadth distal to the tip of the olecranon and one finger's breadth anterior to the dorsal subcutaneous border, the skin over the bone being stretched distally and anteriorly with the operator's finger and thumb (to overcome the slackness of the skin when traction is made later). The elbow should be fully flexed, so that the ulnar nerve is stretched forward and out of the way of possible harm from the wire or pin. The necks of the metacarpal bones are similarly transfixed, one finger's breadth proximal to the knuckles, "Novocain" solution having been injected previously into the interosseous spaces and the inner and outer aspects of the hand over the corresponding necks of the metacarpal bones. To take up the slack on the skin prior to traction, the skin is drawn forward to the palm and proximally. It is advisable to begin transfixion on the ulnar side, as the neck of the index finger metacarpal has a much larger diameter than that of the fifth metacarpal, and is therefore more easily transfixed, should the direction of the wire or pin be not accurately through the centres of the inner metacarpals. The stretched rustless wire method of traction associated with the name of Kirschner, of Tübingen, is now largely replacing Steinman's pin, as the diameter of the wire is between 0.5 and 1.0 millimetre, whereas the pin diameters vary from four to five millimetres and leave a sinus which may be troublesome. The skin round the wire is painted with "Mastisol" ("Benzo-Mastiche", Martindale¹) and a sterile piece of rubber sponge, dabbed with balsam of Peru, is held against the skin by a sterilized collar (Figure VI), so that there is at least 2.5 centimetres (one inch) of sterile wire projecting from the skin on either side. The sticky surface between the balsam and gum gives an anti-septic, elastic, water-tight joint, whereas the gum alone dries hard and stiff, like collodion, and the sponge may peel off later. The wires are now stretched and clamped in the stirrups, which are then harnessed up to the screw traction apparatus designed by Böhler for various difficult fractures of the upper extremity. In a different manner to that illustrated, the apparatus can be used for reduction of fracture-dislocation of the head of the humerus and supracondylar and shaft fractures. Harnessed up as shown, it may be used in the reduction of an old unreduced dislocation of the semilunar bone; the original radio-carpal space is so widened by this powerful method of traction that now the reposition of the dislocated carpal may usually be accomplished.

The forearm is held vertical to assist the blood return and reduce the œdema and hæmatoma; blood and fluid are effused into the synovial sheaths and muscle planes. The forearm is flexed to a right angle, which represents the neutral position; physiological relaxation of the muscles overriding the fracture is thus permitted. This attitude corresponds to that employed in the reduction of fractures of the leg and ankle, when the knee, flexed to 90°, allows relaxation of the calf muscles that are inserted into the tendo Achillis, which in extension is nine centimetres longer. Flexion of the elbow not only permits relaxation of the long flexors arising from the medial epicondyle, but also the extensor group arising from the external epicondyle. The brachioradialis, which, inserted into the styloid process of the radius, with forearm extended, tends to maintain the radial abduction due to the original force, is also now relaxed. The closing power of the fist is diminished by radial abduction, as the hand is normally deviated to the ulnar side; hence the weakness of the hand due to radial abduction following removal of part or whole of the navicular or scaphoid bone for fracture through the neck of the radius. This operation has now become obsolete owing to the wide acceptance of Böhler's

¹ "Mastisol" consists of mastich, 40 parts, castor oil 1.2 parts, benzol to 100 parts. This is filtered. (Naked flames must be avoided.)

Kirschner's apparatus, slightly modified by the author, is obtainable in Melbourne at T. E. Company, 131, Balaclava Road, Caulfield.

teaching that all fresh and the greater number of old fractures, even of two or three years' standing, may be expected to unite if immobilization is maintained long enough (three months to a year). Moreover, in a Colles's fracture radial abduction distorts the bony grooves for the long abductor of the thumb and the *extensor pollicis longus*, which may be frayed by the proximal end of the distal fragment, resulting perhaps some weeks later in spontaneous rupture of the attenuated extensor tendon by some sudden strain or movement. Radial abduction can be controlled mechanically in a stubborn case (for example, a case of some days' standing) by harnessing the hook of the screw traction apparatus to one of the lateral holes of the Kirschner stirrup, instead of the central one, as illustrated. (By such a manoeuvre the abduction deformity in a third degree Pott's fracture can be easily controlled.) Finally, the position of pronation relaxes the *pronator quadratus*.

Gradual screw traction is now used, and over-extension is maintained at first (see Figure III) because thereby the fragments are disentangled from the *pronator quadratus* muscle (in which *myositis ossificans* occasionally develops). The long muscles running to the hand are elongated and stretched as taut straps over the fragments and so act as an encircling band compressing the fragments into normal alignment. The comminuted radius resumes its anatomical outline; the subluxation of the radio-ulnar articulation is reduced; the radio-carpal joint line on the radial side is restored, obviating the later development of osteoarthritis. The wide separation of the articular surfaces diminishes the likelihood of subsequent development of adhesions. Such powerful over-extension applied in the treatment of fractures of the shafts of long bones will always pull out muscle from between the fragments. Indeed, the remarkable manner in which comminuted fragments, even around joints, unless rotated, literally fall into position after skeletal traction has practically excluded operation for closed fresh fractures of shafts of the extremities.

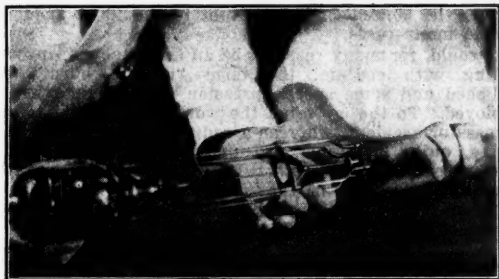


FIGURE V.
Showing Loth electric drill for introducing rustless steel wire. The claw position of the hand causes the metacarpal necks to lie in one plane.

Any slight backward and radial abduction is corrected fairly easily now that the ends are disimpacted and contracted and blood-sodden muscles are stretched; but the slight backward displacement still remaining is better left until the non-padded plaster splint is applied.

The procedures carried out so far should be painless, and when the radial abduction is corrected, pressure on the sensory radial nerve is relieved. A peculiar persistent swelling and bluish shining skin, mainly over the back of the wrist, even met with in the absence of fracture, sometimes follows trauma in this region. X ray examination shows bony atrophy due to haliteresis (decalcification) of the carpus and the distal ends of the radius and ulna. A similar condition may occur in the foot. In the wrist it is due to a trophædema following injury to the gangliform enlargement on the termination of the dorsal interosseous nerve, filaments from which have trophic influences on the bones and carpal joints and distal ends of the radius and ulna. Causalgia may follow. This condition was originally described in connexion with cat bites, the teeth injuring the ganglion.

Oedema and the hæmatoma are now massaged away before a posterior non-padded plaster cast, encircling three-fifths of the wrist, is applied directly to the skin. The splint is made of six layers of slightly wrung out (to facilitate moulding whilst wet) plaster bandage and extends from below the elbow to the heads of the metacarpal bones. It is imperative that free movements at the metacarpo-phalangeal joints remain, otherwise adhesions rapidly develop at these joints and in the tendon sheaths of the wrist and fingers. Böhler has shown that if oedema is carefully massaged away, then a skin tight plaster may be applied which will not become loose in a few days. Moreover, such a plaster cast, moulded exactly to the

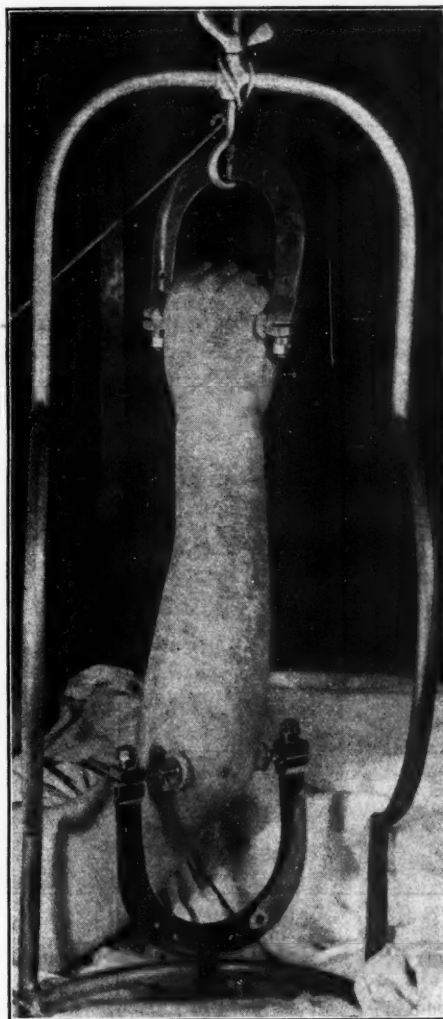


FIGURE VI.
Showing traction and counter traction by Kirschner stirrups, harnessed to Böhler's screw traction apparatus.

subcutaneous bones, obviates the danger of pressure sores, so common with bulky, loosely padded plaster casts, which work loose when the swelling subsides and which never in any way fit the fragments of bone so that they are rendered immobile. The thumb is abducted whilst the splint is being moulded to the dorsum of the hand; otherwise the movement of abduction may be subsequently impaired, due to

adhesions of the tendons of the *abductor pollicis longus* and the extensors of the thumb.

An anterior plaster splint was used in the case herein described, though usually this is not necessary. It should extend to the web of the thumb and overlap the posterior one at the edges; both are now bound on with gauze bandage, wrapped loosely and then covered by a loose circular plaster bandage.

The plaster splint or "slab" method of application obviates the danger of ischæmia and tenosynovitis caused by encircling plaster bandages compressing the limb. It is now important to manipulate the fragments by the fingers or by thenar pressure. In the present case Böhler's *éraseur*, designed for reduction of compression fractures of the *os calcis* or condylar separations, was used as in the illustration (Figure VII). After the plaster has set, or better still, some twenty-four hours later (to prevent the development of œdema by maintaining the vertical suspended position), the apparatus and wires in the bones

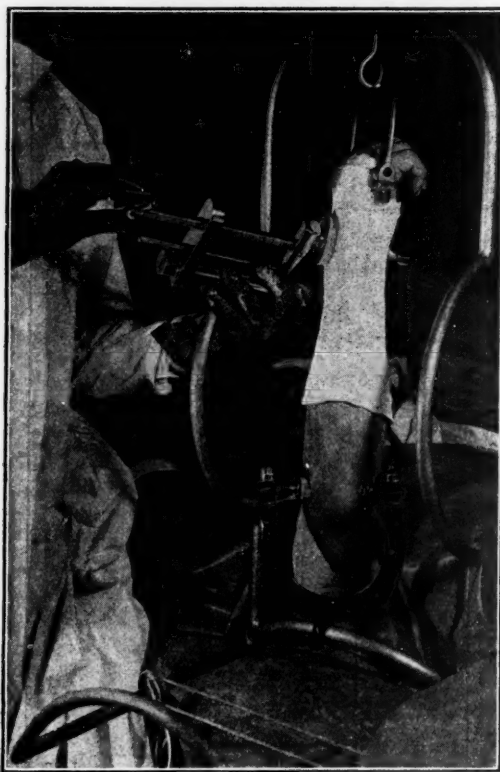


FIGURE VII.

Anterior and posterior plaster splints applied. Until the plaster sets the *pelottes* of the *éraseur* maintain the reduction of the fragments.

are removed—a painless procedure. When, however, such a method is used for controlling the broken ends of both bones of the forearm, the wires remain incorporated in the plaster (the ends being cut off); thus the correctly opposed bone ends are bound up directly by plaster and subsequent slipping eliminated. Usually in such cases, however, the lower ends of the radius and ulna are transfixed instead of the metacarpals.

After-treatment consists in movement of the fingers immediately, and all active movements of the forearm, including pronation and supination, within a day or so. As the bone ends are completely immobilized, active movements of the muscles running over the fracture are permissible. Such movements improve the blood supply

of the limb and prevent troublesome muscle and joint adhesions as sequelæ. Therefore, a sling is avoided at all costs, which may cause a stiff elbow and shoulder also, as these joints are usually concomitantly bruised by the nature of the original violence (for example, falling on the extended hand) and therefore require active movements to avoid adhesions.

Acknowledgement.

For permission to publish this case I should like to thank Mr. Laming Evans, O.B.E., F.R.C.S., Consulting Surgeon to the King Edward Memorial Hospital and Senior Surgeon to the Royal National Orthopædic Hospital, London.

Reviews.

RADIOTHERAPY IN THE TREATMENT OF CANCER.

"RADIUM AND CANCER", by H. S. Souttar, a monograph of sixty-four pages, is eminently suited as an introduction to the study of the nature and therapeutic uses of radium in the treatment of cancer.¹ It is written in a clear and concise style, and its author does not pretend that it is anything more than an introduction.

Chapter I is the longest and most important, and covers in a very clear manner the physics of radium and of the rays and emanations which are thrown off from the parent substance. Chapter II deals with the apparatus and means of applying radium, and with the very important question of screenage. We are quite in accord with the author's opinion that the radium bomb is dangerous. It has not lived up to its reputation of three or four years ago.

On page 28 appears the statement that "rodent ulcer, intractable to all other forms of treatment, vanishes in the presence of radium". Surely this is an inexactitude. Many thousands of rodent ulcers have been, and many others will yet be cured by surgical removal. Nevertheless it must be conceded that radium is the ideal agent for cure in many cases.

It would, for many reasons, be an advantage if, in books dealing with radium, the term "radium burn" were abolished and some such expression as "radium reaction" employed. To the lay mind the word "burn" implies carelessness or accident, whereas a radium reaction is, in some degree, quite inevitable and unavoidable; in fact, it is what is expected. Chapters IV to IX embrace a rather cramped description of the use of radium and radon in seeds upon the malignant growths met with in various organs of the body. Much prominence is rightly given by Dr. Souttar to the advantage of radon emanation, especially in the form of seeds. We agree with his view that special attention should be paid to the peripheral zone of the neoplasm. It is the periphery of a cancer and not so much the centre that kills the patient. Chapter VII deals with carcinoma of the breast. We do not share the author's optimism in regard to the treatment of this common form of malignant disease by radium alone. Cancer of the breast, even with early metastatic axillary deposits, has often been completely cured by radical surgery. The author's views concerning the treatment of cancer of the urinary bladder are also over-optimistic.

The final chapter deals succinctly with untoward accidents that may occur, either to the patient or those handling radium. These accidents are in every sense of the word real.

Throughout the book the author goes to some pains to point out the advantages of using radon seeds when available and when indicated by the needs of the case. With this we agree.

"Radium and Cancer" should be looked upon as purely introductory to the study of radium and not as of value in teaching how radium should be used. To all interested in the study of radium it is heartily commended.

¹ "Pocket Monographs on Practical Medicine: Radium and Cancer", by H. S. Souttar, C.B.E., M.D., M.Ch., F.R.C.S.; 1932. London: John Bale, Sons and Danielsson, Limited. Foolscape 8vo., pp. 64, with illustrations.

ILLUSTRATIONS TO THE ARTICLE BY DR. THOMAS KING.

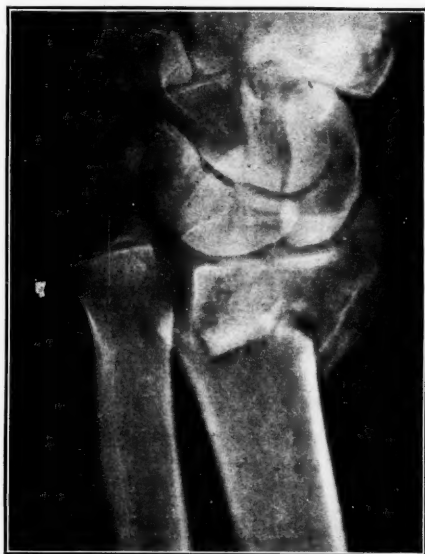


FIGURE I.

Antero-posterior radiogram of five days old Colles's fracture with the distal fragment comminuted and subluxated at the radio-ulnar articulation, before reduction.



FIGURE II.

Lateral view before reduction.



FIGURE III.

Antero-posterior view after screw traction, which has slightly over-extended the fragment. Plaster is applied.



FIGURE IV.

Lateral view after screw traction and the application of a plaster splint.



ILLUSTRATIONS TO THE ARTICLE BY DR. W. J. PENFOLD, HILDRED M. BUTLER AND
DR. I. JEFFREYS WOOD.

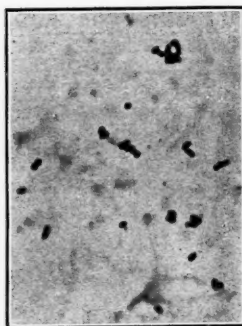


FIGURE I.
Smear from primary culture
of strain "AB" in K medium
after 42 days' incubation.
($\times 1,000$.)



FIGURE II.
Strain "AB" grown on agar
for 16 days. ($\times 1,000$.)



FIGURE III.
Smear from primary culture
of strain "KK" in K medium
after 14 days' incubation.
($\times 1,000$.)

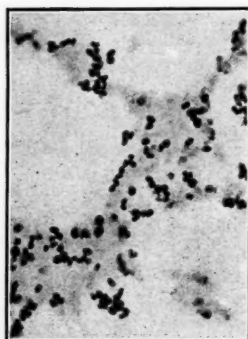


FIGURE IV.
Strain "KK" grown on agar
for seven days. ($\times 1,000$.)

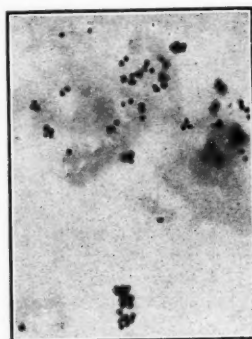


FIGURE V.
Smear from primary culture
of "EC" in K medium after
35 days' incubation. ($\times 1,000$.)



FIGURE VI.
Strain "EC" grown on agar
for ten days. ($\times 1,000$.)



FIGURE VII.
Smear from primary culture
of "EC" in K medium after
35 days' incubation. ($\times 1,000$.)



FIGURE VIII.
Strain "EC" grown on agar
for 12 days. ($\times 1,000$.)

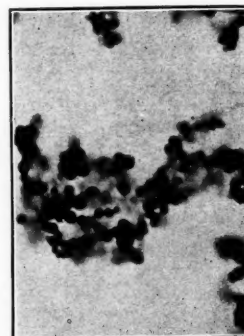


FIGURE IX.
Strain "KK" grown on agar
for 28 days. ($\times 1,000$.)

The Medical Journal of Australia

SATURDAY, JULY 30, 1932.

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References to articles and books should be carefully checked. In a reference the following information should be given without abbreviation: Initials of author, surname of author, full title of article, name of journal, volume, full date (month, day and year), number of the first page of the article. If a reference is made to an abstract of a paper, the name of the original journal, together with that of the journal in which the abstract has appeared, should be given with full date in each instance.

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FOLLOW-UP SYSTEMS.

At the Third Cancer Conference, held at Canberra in March, 1932, Dr. M. J. Holmes gave a résumé of developments which had taken place in connexion with cancer control during the previous twelve months. He gave prominence to the following up of patients at the various centres. He stated that, at the end of a three and a half year period, of 1,265 patients with carcinoma classed as operable, 54% were alive and free of symptoms. Only 4% were known to have died of cancer, but the subsequent history of 23% of the patients was not available. He was careful to point out that in many hospitals satisfactory follow-up systems had been established. At the Melbourne Hospital only 7% of the patients with carcinoma treated since 1928 have not been traced. Those who have attempted to keep in touch with a large number of patients over a period of years will realize that this result is satisfactory. Dr. Holmes gave the figure for Sydney centres as 15%. He said most emphatically that 23% was much too high a figure for those about whom information was not available.

The figures quoted by Dr. Holmes show first of all what can be done in keeping in touch with patients after their discharge from hospital. They also show that, while at institutions such as the Melbourne Hospital the importance of a follow-up

system is recognized, at other hospitals and treatment centres the matter is looked on as of no great importance or is entirely disregarded. This is the more surprising since the figures given by Dr. Holmes refer to patients with malignant disease only. It cannot be argued that a figure of 23% and more, representing failure to trace patients, is due entirely to the indifference of patients or their forgetfulness. It is due to the apathy of hospital authorities and hospital staffs or to the half-hearted administration of a badly organized department. When a medical practitioner undertakes the treatment of a patient with malignant disease, his responsibility does not end when he has removed, irradiated, cauterized or otherwise treated the original growth, and when he has irradiated or attempted to remove any palpable secondary deposits. He must not wait until the patient is compelled to seek advice for secondary manifestations. It is of little use relying solely on a patient's observation of himself. Continuous supervision is necessary from the patient's point of view for the discovery of recurrences and also that the comparative value of various methods of treatment may be determined. As far as Dr. Holmes's figures are concerned, it is not an exaggeration to state that when no attempt, or only a half-hearted attempt, was made to keep in touch with cancer patients, the workers have been guilty of a breach of trust with those who supplied the means of treatment and they have not done their duty to patients.

Malignant disease is not the only condition in which the following up of patients after their discharge is invaluable. In pulmonary tuberculosis; heart disease; diabetes; nephritis; gastric ulcer, especially when the patient has been subjected to gastro-enterostomy; in mental and in other conditions, treatment does not end when the patient is sent home. Certain acute infections, unaccompanied by complications, are perhaps the only conditions in which it is not necessary for the patient to be kept under supervision after his discharge. Present-day hospitals are not mere hostels in which the patient is tided over the acute stages of an illness. They should be regarded as centres from which his subsequent health is controlled. They are also, of course, centres in which disease should be

studied in its incidence and progress, if necessary over long periods. Medical practitioners, if they attempt to do their duty by their private patients, do not allow those suffering, for example, from nephritis, heart disease or pulmonary tuberculosis, to live, year in, year out, without supervision. Why should they adopt slipshod methods with their hospital patients? The responsibility in each instance is the same.

Hospital boards must have this matter brought to their notice by the medical staffs. The boards may hesitate at the prospect of having to employ a few clerks to control such a system, but if they realize that in existing circumstances they are not doing justice to their patients, they will perhaps think otherwise. A follow-up system is bound up with the keeping of adequate records and ought to be used in conjunction with a system of hospital almoners. In some places nurses are engaged in visiting the homes of tuberculous patients, and in these areas the adoption of a complete system should not be difficult. The work can be done. The Melbourne Hospital is doing it with malignant disease, and doing it well; this example should be followed.

Current Comment.

ATELECTASIS IN THE NEW-BORN.

At one time the term *atelectasis pulmonum* signified a condition of the lungs in which complete expansion had not occurred, the condition being partial or total. The term collapse of the lung indicated a return, partial or complete, of expanded and aerated lung to the foetal state. This distinction is not now maintained. As originally defined, atelectasis is congenital and seen in weakly infants whose respiratory movements do not suffice to draw in air to the necessary amount. The child's body may be otherwise healthy and the lungs in a normal state, readily expanded by artificial introduction of air. Atelectasis may be the cause of death, not only in weakly and immature infants, but also in otherwise strong and healthy babies. Atelectasis may sometimes be explained by arrest of the placental circulation during labour—the flow of blood to the child through the umbilical cord is obstructed, and death may occur before the establishment of pulmonary inspiration. The child is born asphyxiated. Every uterine contraction interrupts “placental respiration” and, if the action of the placenta be destroyed before the onset of respiration, atelectasis may result. The bodies of atelec-

tatic infants may present serious medico-legal problems. The value of X rays in diagnosis of pulmonary conditions in the new-born is shown in two recent articles.

Ethel C. Dunham has investigated atelectasis in the new-born.¹ She observes that this condition is the abnormal persistence of incomplete expansion of the lungs, which is physiological in the first few days of life. With the onset of respiration expansion begins, but generally is not complete for some days. During the first few days of life some atelectasis is physiological but symptomless; when symptoms occur, it is pathological. The commonest cause of persistence of the foetal state in parts of the lungs is held to be failure of proper functioning of the respiratory centre. Sometimes the cause of such failure cannot be ascertained; immaturity, asphyxia, cerebral hæmorrhage or œdema may be responsible. Or mechanical factors may be the cause, such as plugging of a bronchus by mucus or pressure on the lung by an enlarged heart or thymus. Atelectasis of the new-born is always secondary to a condition interfering with the mechanism causing full expansion of the lungs.

Dunham reports six cases of new-born infants in whom the diagnosis of pathological atelectasis was confirmed by X ray examination. As a routine, carbon dioxide (5%) with oxygen (95%) was administered with only one fatality at the age of thirty hours. The autopsy disclosed a patent *ductus arteriosus*, which seemed about the same size as the aorta. The right and left pulmonary arteries were patent and of usual size. The ventricles and valves of the heart were normal. There was no enlargement of the heart, though the X rays seemed to suggest that enlargement was present. The left lung was dark red, firm and non-crepitant; it was typically atelectatic. All the infants were of or above average birth weight. Labour was “prolonged” in one case and “difficult” in another. In four the infants were delivered spontaneously, and in two by operative measures. Cyanosis was present in all. Only one had a cough; in three the cry was abnormal, and in five respirations were abnormal. Lung signs were noted in four. Râles were present in two. In three the percussion note was impaired over a localized area; in two of these the breath sounds were altered over this area (diminished in one and tubular in the other). Physical signs varied from time to time. In two were no physical signs, and the diagnosis was made from symptoms corroborated by X ray examination. In X ray examination the lung fields may show a general haziness, due to lack of complete aeration (previously wrongly interpreted as due to faulty technique). Or one or more areas of increased density may be seen. Or one lung may show cloudiness of greater or less intensity while the other is clear. If one lung be involved more than the other, there may be a difference in the level of the diaphragm, the affected side not descending as well as the other in inspiration. If the atelectatic area

¹ American Journal of Diseases of Children, March, 1932.

be in the left paravertebral region, the pulmonary change will be obscured by the heart shadow and may not show in the films. X ray examination should always be made in a new-born cyanotic infant. C. A. Weymuller, A. L. L. Bell and A. A. Trivilino report a daily radiological study of fifteen normal premature babies during the first fourteen days of life.¹ Pneumonia of the new-born, they state, is known to occur, but seldom recognized. X ray examination of the thorax is of the greatest help in diagnosis in infants with unexplained cyanosis or disturbances of the respiratory rate or rhythm from varying degrees of collapse. In the series recorded, if cyanosis existed, or feeble cry, dyspnoea, excessive loss of weight or other abnormality, the films were excluded. One infant, cyanotic at birth, was resuscitated artificially and was normal at subsequent examinations. In no case was there X ray evidence of atelectasis, though the infants should have been promising subjects for such.

In this connexion reference may be made to the view of P. N. Coryllos and G. L. Birnbaum, that pneumococcal pneumonia is an atelectasis. Their work has previously been discussed in these columns. They bring evidence showing that the size of the consolidated lobe in pneumonia is smaller than normal, and the seeming enlargement of the consolidated lung is due to collapse of the healthy lung when the thorax is opened at autopsy. They consider that there is only one determining cause of post-operative collapse or atelectasis, that is, complete bronchial obstruction, and that post-operative collapse and post-operative pneumonia are but phases of the same morbid condition. W. E. Lee, G. Tucker and L. Clerf think that two factors are always present in post-operative atelectasis—a thick, viscid bronchial secretion and inhibition of coughing. The tenacious sputum accumulates in the dependent parts of the bronchial tree and at one or more points the lumen is completely occluded and lobular or massive atelectasis results.

THE GALL-BLADDER AND CARDIAC PAIN.

THAT region of the body lying near the mid-line and extending a little above and below the diaphragm is well known as a storm centre from both symptomatic and diagnostic points of view. There are several acute emergencies that may arise in this position, and differentiation between them is not easy.

For instance, it is well recognized that coronary occlusion, acute cholecystitis and ruptured ulcer may present features of similarity. Perhaps less widely realized is the fact that gall-bladder disease is not only common in association with cardiovascular crises, but may actually suggest a causal relation therewith. C. H. Miller² has reviewed this association in a brief but interesting communica-

tion, and appends some illustrative cases. He apologizes for the term "cardiac pain", but cannot suggest a better; he remarks: "Everyone knows what you mean." The term may have the merit of being a general one; the pain referred to is not limited to any special anatomical district and is not due to a narrowly defined lesion. If the term is used in a general sense, it need not commit us to assuming that the cause of the pain is actually in the heart. It would sometimes be more satisfactory to use the term precordial pain.

The *a priori* reasons for considering the lesions of the gall-bladder of importance in the causation of so-called cardiac pain are succinctly stated by Miller as follows. The gall-bladder may be a septic focus; it may contain stones, which may be responsible for attacks of severe colic; there may be some disorder of chemistry which could perhaps lead to defects of circulation; the innervation of heart and gall-bladder is from neighbouring regions of the central nervous system.

Several case histories are given illustrating syndromes indistinguishable from *angina pectoris*, and in which the capacity for cardiac effort was very limited owing to the severe attacks of pain induced thereby. In each case cholecystectomy gave relief from symptoms. Other cases illustrate the occurrence of pathological states of both the heart and gall-bladder in the same subject.

The next evidence brought forward is an interesting statistical survey of three hundred and fifty cases of cholecystitis and gall-stones studied *post mortem* in the London Hospital during the last ten years, with particular reference to the frequency and severity of atheroma. Professor Major Greenwood has impartially summed up these figures, and considers that there is a definite evidence that the incidence of atheroma in these persons was too high to be accounted for by the usual causes alone; in other words, that there appears to be an association between the arterial degeneration and the gall-bladder disease, particularly in the age groups fifty-one to sixty and sixty-one to seventy, in both males and females, and to a less extent forty-one to fifty in men.

These points emerge. Gall-bladder pain and true cardiac pain are difficult to distinguish, and may coexist. There is evidence that the subjects of gall-bladder disease are rather more prone to suffer from atheroma than others. Cases are recognized in which removal of the gall-bladder not only gave relief from pain that might have been thought to arise from either the gall-bladder or the heart, but has actually improved the capacity of the heart to respond to effort without rebellion. It is also pointed out that so-called cardiac pain is not of necessity a contraindication to operation. The last statement leads us to the practical consideration that where a patient suffers from undoubted gall-bladder disease and also cardiac symptoms, even where there is doubt as to whether the latter are due to the former or not, he need not be denied the relief that the surgeon can bring.

¹ American Journal of Diseases of Children, March, 1932.

² The Lancet, April 9, 1932.

Abstracts from Current Medical Literature.

BACTERIOLOGY AND IMMUNOLOGY.

Yellow Fever.

THE duration of the passive immunity of monkeys to yellow fever has been studied by Johannes H. Bauer (*The American Journal of Tropical Medicine*, November, 1931). He prepared a pooled serum obtained from four monkeys that had recovered from yellow fever. Monkeys to which this serum was administered remained completely immune for three weeks, and were partially immune for seven weeks. Immunity was then lost. A pooled serum was obtained from seven African natives who had recovered from yellow fever. Varying quantities of this serum were injected into monkeys which were then inoculated with yellow fever virus. It was found that an injection of three cubic centimetres or more of the immune serum protected monkeys against yellow fever; an injection of two cubic centimetres conferred immunity on one of two monkeys and partial immunity to the other; an injection of one cubic centimetre conferred full immunity on one of two monkeys, and apparently none at all on the other. It was found that the administration of human immune serum in an adequate dosage protected monkeys fully for seven days. Monkeys inoculated with yellow fever virus after the expiry of fourteen days or more from the time the serum was administered, all died of yellow fever. The author makes the suggestion that, by analogy, passive immunity conferred on man by the administration of human convalescent serum (if immunity can be so conferred) should last longer than if the serum of monkeys or other animals were employed. He remarks further that "as human serum does not sensitize man, its use has a distinct advantage over that of other species for repeated injections at short intervals for prophylactic purposes".

Cellular Reactions to Fractions Isolated from Tubercle Bacilli.

F. R. SABIN (*Physiological Reviews*, April, 1932) has studied the cellular reactions to chemical fractions from the tubercle bacillus, and has shown that there are three different types of complex lipids in the organism which can be discriminated by the cellular reactions they produce. The cellular reactions to the tuberculo-phosphatide were first studied and it was found that this substance reproduced the tubercle; it was phagocytized by the monocytes of the connective tissue and partially degraded within them, the epithelioid cell being thereby formed. Caseation is believed by the author to be the end stage of the epithelioid cell, and the infiltration of leucocytes to be secondary to the death of these

cells. Characteristic myelin figures of the phosphatide could be seen within the living cell, indicating that it was the substance itself and not some contaminating impurity which was responsible for the reaction. The tuberculo-phosphatide was found to act as an antigen. The only constituent of the phosphatide which could produce the typical cellular reaction was a saturated fatty acid of high molecular weight, phthioic acid. All the other lipids, the wax and the acetone soluble fat, were found to contain some of this phthioic acid and to possess varying degrees of specific biological activity. The waxes contained phthioic acid and an unsaponifiable residue, which was shown to be a higher alcohol, and this residue, in spite of its insolubility in water, when injected in an oil, acted as a remarkable stimulant toward the production of undifferentiated connective tissue cells. It was always irritating and called leucocytes from the vessels. The most complicated cellular reactions were produced in response to the acetone soluble fat, and supravital studies showed that every type of connective tissue cell had been stimulated, which may be due to the fact that the acetone soluble material is a complex mixture of fatty acids. The characteristic cellular response to tuberculo-protein was found to be the plasma cell, and the tuberculo-polysaccharides were chemotactic and toxic to neutrophilic leucocytes.

Atypical Lactose-Fermenting Organisms.

J. A. KENNEDY, P. L. CUMMINGS AND N. M. MORROW (*Journal of Infectious Diseases*, April, 1932) report the recovery of atypical lactose-fermenting organisms in the routine examination of stools and urine from patients with intestinal toxæmia, nephritis, pyelitis, typhoid fever and from suspected "carriers". The material examined was plated on Endo's medium, and a group of twenty-two strains which yielded colourless colonies was recovered. On transplantation to lactose broth, acid and gas were produced in from two to fourteen days. All the organisms were Gram-negative, non-sporing bacilli; twelve were motile and ten non-motile. Twenty-one produced indol, but its production was delayed for from two to six days, so was not noticed at first. All reduced nitrates to nitrites, failed to liquefy gelatin within fourteen days, and were Voges-Proskauer negative and methyl-red positive. Other cultural and biochemical reactions were carefully studied. The authors consider that these organisms probably occupy a position intermediate between the lactose-fermenting colon bacillus group and the non-lactose-fermenting paratyphoid group as regards pathogenicity and cultural characteristics. The fact that twenty-one out of twenty-two strains produced indol, and that by daily transfer of the organism from lactose broth to fresh lactose broth a speeding up of the rate of

fermentation could be secured would seem to link them closer to the colon bacillus than to the paratyphoid group. It is suggested that when non-lactose-fermenting colonies, isolated on various media for recovery of enteric organisms, fail to allow of classification, information may be gained by incubation of the lactose broth cultures for at least fourteen days.

Inoculation with Typhoid Vaccine.

ROY F. FEEMSTER (*Journal of Infectious Diseases*, February, 1932) examined a group of ninety students with a view to ascertaining antibody response to inoculation with typhoid vaccine in those previously inoculated, those who had suffered from an attack of typhoid fever, and those who had had neither. He summarizes his results as follows: "Persons who have had typhoid fever or who have had inoculations with typhoid vaccine produce larger amounts of antibodies in response to subsequent inoculations with typhoid vaccine than those who have not had typhoid fever or been vaccinated against the disease. The routine injection of three doses of typhoid vaccine may not give as high a degree of immunity as has been believed. A second or third injection some weeks or months after the first would seem to be indicated. The Widal reaction is less reliable in the case of a person who has been vaccinated two or more times than in that of a person who has been vaccinated only once. The severity of the reaction to the injection of typhoid vaccine is in no way related to the immunity that is formed in response to the injection."

Respiratory Flora of Eskimos.

J. R. WELLS AND P. HEINBECKER (*Journal of Infectious Diseases*, April, 1932) studied material consisting of direct smears and cultures from throats of one hundred and fifteen persons living within the Arctic Circle and representing seven isolated groups of Eskimos. The general conditions of health among the various groups at the time of the examination were good and no acute respiratory infections were evident. There had been no contact with the outside world for a year or more in most cases and all material was collected as soon as the vessel dropped anchor and before contact with members of the crew had occurred. The results showed that in general the respiratory flora of these people is very similar to that of persons living elsewhere. The most abundant organisms found belong to the group of staphylococci, many of which were of the hæmolytic variety, and to the chromogenic Gram-negative diplococci. Other groups of organisms found, in order of frequency of occurrence, were *Streptococcus viridans*, diphtheria-like bacilli, sarcinæ, *Micrococcus pharyngis-siccus*, *Micrococcus catarrhalis*, *Micrococcus tetragenus*, *Streptococcus hæmolyticus*, with Friedländer's bacillus, strepto-

thrix and yeasts less common. The examination of direct smears revealed the presence of many fusiform bacilli and spirochaetes apparently of the same type as those usually found in the mouths of most people.

HYGIENE.

Medical Supervision of Air Ports.

OSCAR M. HOLDEN (*Journal of State Medicine*, August, 1931) points out the effect of air-borne traffic in bringing countries infected with dangerous diseases into closer contact with those that are uninfected. Modification of quarantine measures is necessary. Medical inspection is an insufficient safeguard, and the principle of requiring medical certificates of physical fitness, viewed at the port of departure, combined with surveillance of all passengers from infected areas, appears more reliable. Certain aerodromes should be specified "Customs Aerodromes" and medical officers should be attached to these. Isolation hospital facilities should also be available. In the event of forced landing, passengers and crew should not be permitted to depart except as necessary for shelter, safety or treatment, and should be conveyed to the nearest customs aerodrome. With proper cooperation between the local health authorities and the aerodrome, bills of health are not necessary. Aerodromes situated in yellow fever zones should be protected against mosquitoes. Intercommunication between medical officers on long distance air routes is valuable. Inspection and fumigation of aircraft by hydrocyanic gas is necessary, and proper certificates should be issued.

The Carcinogenic Potency of Mineral Oils.

C. C. AND J. M. TWORT (*Journal of Industrial Hygiene*, August, 1931) describe the results of applying certain mineral oils to the backs of one hundred mice for a considerable period of time in an endeavour to estimate the cancer-producing ability of the agents. Carcinogenic potency varies with the concentration of the agent, the amount applied, the nature of the diluent used, the area covered by applications, the number and frequency of applications, the susceptibility and health of the animal, the greasiness of the skin, hygienic and dietetic conditions, and, possibly, the season of the year. The potency of various oils is estimated by a mathematical process depending on the time in weeks taken to produce a certain number of tumours in animals; in other words, the response of the animals to the carcinogenic agents is expressed as a function of time. Refined Scotch shale lubricating oil was found in every instance to be more potent than any refined petroleum well oil, and the most potent fraction was found to be the unfinished

lubricating oil. The samples of refined petroleum tested were found to vary considerably in potency, and as a rule the heavier grades were found to be more potent than the lighter. The addition of lanolin or sperm oil lessened the carcinogenic potency. The fluorescence of oils may be related to their potency. Chrysene has a definite carcinogenic potency.

The Prevention and Control of Leprosy.

R. G. COCHRANE (*Journal of State Medicine*, October, 1931), in stressing the value of a knowledge of the bacteriology and pathology of a disease when considering prophylaxis, discusses heredity, age, contagiousness and immunity in relation to the prevention of leprosy. Although latent infection may develop in the apparently healthy children of leprosy parents, this is rare, and no other evidence exists in favour of leprosy being an hereditary disease. On the other hand, age is probably one of the most important aetiological factors, the most likely age for acquiring the disease being from fifteen to thirty years. Children are very susceptible. The examination of children should form a part in any preventive scheme. The degree of contagiousness is the most important factor in any such scheme. At present this question is unsettled. At the International Leprosy Conference at Manila, "open" and "closed" types of the disease were discussed. The contagiousness may be enhanced by other factors, such as the individual, race and climate. The presence of other chronic diseases, vitamin deficiency and bad diet and social conditions probably increase susceptibility. The examination of contacts is important. Humid climate is probably a factor in the spread of the disease. It has been generally assumed that toxins of leprosy as such do not exist, and no antibody has been discovered; consequently there is no evidence of individual immunity. On the other hand, racial immunity does exist, and the different types of leprosy predominate among different races and in different areas. A system for the control of leprosy should include a survey of the population; facilities for treatment of early symptoms and isolation and treatment of "open" cases, examination of contacts, examination of school children and immigrants, training of medical officers and dressers, and propaganda. As long as control measures are based on lack of knowledge or the refusal to apply modern preventive principles they will be ineffective.

Vasomotor Reactions to Localized Drafts.

C. E. A. WINSLOW AND L. GREENBURG (*American Journal of Hygiene*, January, 1932) have conducted twenty-six experiments on seven students in an ordinary laboratory room with controllable atmospheric conditions. The air temperatures ranged between 8.3° and 28.3° C. (47° and 83° F.), relative

humidities between 19% and 69%, and air velocities between 4 and 10 metres per minute. The subjects wore only a pair of short trunks and reclined on a cot. After forty-five to sixty minutes observations were made of the dry and wet bulb temperature; kata thermometer; Glatzel mirror observations of nose and throat were made; pulse, respiration and blood pressure and the subject's comfort were noted; and temperatures of the skin, the nose and throat were taken. The head or feet of the subjects were then isolated by a yoke of beaver board and the extremity only was subject to the draught of air produced by an electric fan, giving a velocity of 120 to 410 metres per minute for thirty to fifty minutes. After a further period of fifteen to fifty minutes the draught was discontinued and a further set of observations were made. A full discussion of the value of the respective methods of observation is given, including a valuation of the moisture deposit on the Glatzel plate. The temperature of the oral mucosa is far more stable than that of the nasal mucosa, varying only from 33° C. in the coldest to 34.5° C. in the warmest atmosphere studied. The temperature of the nasal mucosa is nearly 3° below that of the forehead and cheeks, while that of the oral mucosa is about 1° higher than that of the forehead and cheeks, the temperature varying in the nasal mucosa from 25° to 34° C. approximately. Chilling of the body causes ischaemia and not hyperaemia of the mucous membranes. The localized draught on the head caused a definite fall in temperature, especially of the nasal mucosa, vasoconstriction being less when the rest of the body was at a higher effective temperature than when the whole of the body was affected. The head recovered its normal temperature very rapidly after cessation of a local draught. It was noted that distinct reflex vasomotor changes also occurred in the hands and feet, with little effect elsewhere. On the feet a localized draught caused a definite and sharp fall in skin temperature, followed by a slow recovery without any marked or constant reflex changes elsewhere, except in the temperature of the nasal mucosa and a less pronounced reflex rise in the temperature of the oral mucosa. Localized chilling of the head caused a fall in the surface temperature of the nasal and oral mucosa of 1° to 3.7° C., with slow recovery. One subject showed exceptional responses. None indicated that localized draught produced vasomotor derangements differing from those caused by uniform chilling of the body surfaces as to suggest a special influence on respiratory infection. Chilling of the head caused less vasoconstriction of the mucous membranes than exposure of the entire body, and chilling of the feet actually caused dilatation of the blood vessels of the nose and throat. The authors do not consider the general belief that chilling of the feet is specially harmful as confirmed.

British Medical Association News.

SCIENTIFIC.

A MEETING OF THE SECTION OF MEDICINE OF THE NEW SOUTH WALES BRANCH OF THE BRITISH MEDICAL ASSOCIATION was held at the British Medical Association House, 135, Macquarie Street, Sydney, on June 16, 1932, Dr. C. B. BLACKBURN, the President, in the chair.

Chronic Diarrhœa.

On behalf of Dr. Allan S. Walker, Dr. E. H. Stokes read a paper entitled: "Chronic Diarrhœas: Their Ætiology and Treatment" (see page 136).

Dr. M. C. LIDWILL expressed his appreciation of Dr. Walker's very comprehensive paper, but he remarked that he did not agree with some of Dr. Walker's statements. Dr. Walker had remarked that only one case of sprue had been observed at the Royal Prince Alfred Hospital during the period in which his investigations were conducted. When Dr. Lidwill was engaged in out-patient work, he had seen seven patients suffering from sprue, five at the Royal Prince Alfred Hospital and two at the Mater Misericordiæ Hospital. When travelling in Queensland four years ago, he had been informed that some 20% of people residing in the Wide Bay district suffered from sprue. It had been observed that only those people who drank jungle water became affected with the disease.

Dr. Lidwill did not believe that people suffering from muco-membranous colitis were primarily neurotic. He remarked that if one were continually to sit on a pin or to suffer from toothache, one would become neurotic. He believed that there was always an organic basis for this condition. Many years ago Dr. Foreman, at his request, had operated on several patients and had found the colon banded down in the sigmoid region. He had adjusted this abnormality after considerable difficulty and the patients had remained well thereafter. Women who had borne children and had suffered some pelvic damage were the most frequently affected with muco-membranous colitis. In the treatment of this condition he employed castor oil every morning for many months and prescribed von Noorden's anti-constipation diet.

In the treatment of ulcerative colitis Dr. Lidwill placed the patient in the knee-chest position and washed out the bowel with half a gallon or even a gallon of a 1 in 10,000 solution of silver nitrate twice a day for ten days. During the following fortnight the bowel was washed out once a day and the strength of the solution was gradually increased to 1 in 5,000. This solution was employed every day thereafter as long as necessary. Within two months the patients were generally very much better. Dr. Lidwill insisted on the necessity for an absolute milk diet during the period of treatment, if necessary for months. Dr. Lidwill thought that he was the first in Australia to use kaolin, as he had read of its value in the treatment of cholera in China. He had found it quite useless in the treatment of ulcerative colitis, but of some value in tuberculous enteritis.

Dr. A. W. HOLMES & COURT remarked that the subject was so vast that it was difficult to discuss. First he proposed to make some remarks concerning amœbic dysentery. Dr. Walker had stated that the cases in Sydney were mostly importations. But within recent years, at Sydney Hospital, eight patients had received attention who had never been out of the State. In the treatment of amœbic dysentery "Yatren 105" had been hailed as a therapeutic agent of the greatest value. He had employed it in addition to emetine. His experience was that "Yatren" occasionally gave brilliant results, but for the most part was not so uniformly satisfactory.

Dr. Walker's experience in regard to Bargen's diplococcus had been similar to his own. For a long time he had been accustomed to ask the bacteriologists to search for Bargen's organism, but as yet they had been unable to isolate it. Dr. Holmes & Court mentioned that the origin of infection by Bargen's diplococcus was said to be in areas of sepsis at the apices of teeth. He found this

difficult to prove. Streptococci frequently appeared to be a causal agent in ulcerative colitis. In a recent case all forms of treatment, including lavage and vaccine therapy, had failed. Besredka's antiviral applied locally had been followed by immediate improvement. Dr. Holmes & Court had no doubt that the antiviral had been a therapeutic agent of great value in this case. He referred to Hurst's method of administering large doses of dysenteric antiserum intravenously. It was said that if the injection was followed by severe anaphylactic shock there was a good chance of improvement. In his own experience the shock had sometimes been alarming and he confessed to having nearly killed two people by the intravenous injection of the serum.

The statement had been made that radiological methods of diagnosis had reached a high state of accuracy. No doubt this was true, but he pointed out the frequency with which advanced malignant disease of the colon remained undiagnosed by radiological means.

Dr. Holmes & Court then referred to a woman suffering from Addison's disease, shown by Dr. Walker at a recent clinical meeting at the Royal Prince Alfred Hospital. This patient had been admitted later to the Royal Hospital for Women. Cortical adrenal hormone ("Eschatin") had been administered and had produced the most striking therapeutic effect. The vomiting and diarrhœa had ceased almost immediately.

Dr. Holmes & Court remarked that he found ulcerative colitis a very puzzling condition. There seemed to be no regularity in the progress of the disease nor in its response to accepted remedies.

Dr. O. A. DIETHELM remarked that all present should be deeply indebted to Dr. Walker for his comprehensive survey of the subject of the diarrhœas. Dr. Walker's paper was of all the greater importance as it contained such a wealth of clinical material. Reference had been made to the occurrence of amœbic dysentery in persons who had never been out of the State of New South Wales. Recently he had seen two such cases himself. Another of his patients had suffered from typical amœbic dysentery and had later developed malignant disease of the sigmoid colon, of which he had died. The condition had been demonstrated *post mortem*. Dr. Holmes & Court had raised the question of the value of "Yatren 105". Undoubtedly this drug was of tremendous value when used in conjunction with emetine. Dr. Diethelm had noticed that its effect was greatest when it was retained in the bowel for some hours. In some cases, however, "Yatren" had been disappointing. When he had been in the United States of America, the treatment of amœbic dysentery had been discussed, and he had heard one man claim the most remarkable results for treatment with injections of fluorin followed by the application of X rays.

Dr. Diethelm had also observed good results from local application of Besredka's antiviral in the treatment of ulcerative colitis.

Dr. Diethelm remarked that in many instances so-called mucous colitis (this term was loosely applied to many conditions) was often associated with some infective process. In some instances it was undoubtedly due to food allergy. He recalled one particular case in which intradermal tests of food extracts had been conducted and the food responsible for the colitis had been eliminated from the diet, with prompt improvement.

Of the various irrigations he had had more success with "Neutral Acriflavine" than with anything else. It was supposed to be quite harmless and non-toxic, but on two occasions in his experience it had caused some slight general disturbance. Surgical treatment had received some mention. He had seen two patients for whom colectomy had been performed with appalling results. He was afraid that he could work up no enthusiasm for surgery in these cases.

Dr. Walker had mentioned "cathartic" colitis. Undoubtedly this condition was commonly met with in practice, and was regulated by a close supervision of aperients and diet in most cases.

Dr. LAURENCE HUGHES expressed his thanks to Dr. Walker for his excellent paper and for the masterly manner

in which he had handled a difficult subject. He mentioned that in tropical countries amœbic and bacillary dysentery were not uncommonly associated, and when the amœbic form dominated the picture the bacillary factor was apt to be overlooked; in such instances a chronic ulcerative colitis was likely to follow as a sequel to the bacillary infection. Hurst contended that chronic ulcerative colitis was a form of bacillary dysentery; in this regard it was worthy of note that, although dysenteric diarrhœa was not an uncommon complaint of children, chronic ulcerative colitis was comparatively rare. On the other hand, one not infrequently observed children suffering from chronic catarrhal colitis; these children were not sufficiently ill to be admitted to hospital, and were more commonly seen in out-patient departments. They often had an associated history of chronic constipation; the chemical and mechanical irritation of the lower bowel produced by this condition and by the injudicious use of aperients gave rise to a colitis. Chronic catarrhal colitis was commonly associated with nervous symptoms. Dr. Hughes suggested that there was a tendency for the term "colitis" to be used too loosely; it might well be reserved for those conditions in which a definite inflammation of the colon existed.

In regard to the treatment of chronic ulcerative colitis, Dr. Hughes stated that his experience had been mainly limited to that employed on service in the tropics. Concerning the local treatment of amœbic dysentery, he had obtained satisfactory results from enemata of quinine solution (1 in 5,000).

Dr. R. S. STEEL remarked that Dr. Walker was to be congratulated on his paper, which was so comprehensive that it was difficult to think of anything that had been missed. Dr. Steel thought that he had found one condition that Dr. Walker had not mentioned, namely, pellagra. In this disease diarrhœa was an important symptom.

Dr. Steel stressed the importance of irritation by food in the causation of diarrhœa. The common errors of taking food containing much waste were largely responsible for many cases of chronic diarrhœa. In a recent paper Alvarez recorded the answers to a *questionnaire* concerning the effects of "roughage" that he had sent to 400 medical practitioners in the United States of America. The general belief of these men was that the value of "roughage" was greatly over-rated and that it caused greater gastro-intestinal trouble than could be feared from a lack of vitamins.

Many people suffering from "nervous" disorders of the bowel became more and more neurotic, and medical practitioners must be very careful not to draw undue attention to the bowel. Dr. Steel felt that constant consideration of bowel disturbance was a common source of increased neurosis.

There was no satisfactory evidence, in his opinion, for placing mucous colitis in the list of allergic diseases. Dr. Walker had obtained a good result from the therapeutic use of urinary protease in one case, but Dr. Steel believed that this result was of no value. He had investigated a number of cases with a view to finding evidence of allergy and had been disappointed. He believed that a positive skin reaction to protease was no proof that a person was allergic. He mentioned that he had tested himself with four different materials and that a reaction had occurred in each instance. Nevertheless he had never had any symptom of an allergic disorder.

Dr. E. H. STOKES expressed his thanks for Dr. Walker's paper. There had been one or two cases in his own experience which might be worthy of notice. Recently, in the Sydney Hospital medical out-patient department, he had seen two cases of amœbic dysentery. The patients were children who had never been out of Sydney. Another patient under his care in Sydney Hospital had suffered from abscess of the lung. No amœbæ had been found in the stools or pus from the lung, but administration of emetine had been followed by recovery. Dr. Stokes had employed Hurst's method of treatment in one case. A dose of ten cubic centimetres had been administered intravenously at the commencement of the treatment and had been increased until it had reached eighty cubic centi-

metres. At this stage the resident medical officer had upset the phial containing the last of the antiserum. That evening the patient had become affected with a severe urticarial rash. She had later recovered sufficiently to be discharged from hospital. She had attributed her recovery to the resident medical officer's clumsiness. When seen some twelve months later this patient had declared that she was now quite well, having cured herself with a fermented decoction of prickly pear.

Dr. Stokes referred to the occurrence of cancer of the bowel. He had recently seen a patient at Sydney Hospital who had been thought to be suffering from dysentery and whose feces had been examined in the pathological department. No amœbæ or dysentery bacilli, however, had been found. The patient had not been seen again until six weeks later, when examination had revealed a mass in the sigmoid region, which had proved to be cancerous.

Dr. Stokes also stressed the neurotic element in mucous membranous colitis, and quoted an illustrative case.

Dr. KEMPSON MADDOX also expressed his thanks and remarked that Dr. Walker had treated a difficult subject in his characteristically efficient manner. Dr. Maddox mentioned the diarrhœa which occurred in the late stages of myeloid leuchæmia. He had recently seen a patient whose spleen was enlarged and whose blood count was that of myeloid leuchæmia in the aleuchæmic phase. The patient had died shortly afterwards and a malignant growth had been found in the colon. The spleen had also been involved in malignant disease. Dr. Maddox asked whether it had been the experience of others that diarrhœa was a common symptom in myeloid leuchæmia.

Dr. Maddox also discussed the diarrhœa associated with achlorhydria. In the treatment of this condition he administered large doses of dilute hydrochloric acid, to be sipped during a meal and for two hours afterwards. He had recently treated with success a patient suffering from the milder form of ulcerative colitis. He had administered a mixture of bismuth and chalk and had given acid alternately with the mixture. In addition, he had employed lavage with a solution of hydrogen peroxide (1 in 80). He had been interested to note later that the use of hydrogen peroxide was recommended in the "Medical Annual" for 1932. He had used Tidy's method of treatment in two cases and in neither had the patient been able to tolerate the silver salt. He thought that as the disease was apt to clear up spontaneously, people must be chary of ascribing any good results to whatever treatment was employed.

In conclusion, Dr. Maddox remarked that of the parasitic forms of diarrhœa possibly the uncinal was more common in Australia than the bilharzial.

Dr. A. B. LILLEY remarked that he had arrived too late to hear the paper, but he had been very interested in the discussion, particularly in regard to the bacteriology of ulcerative colitis. He had searched for Bargen's diplococcus in two cases by the actual swabbing of the ulcers through a sigmoidoscope and had been able to isolate an organism fulfilling most of the cultural requirements of this diplococcus. Possibly the reason why it was seldom found was that it was seldom looked for. Dr. Lilley also mentioned the frequency with which late lactose fermenting bacilli were found.

Dr. C. B. BLACKBURN said that he was sorry that Dr. Walker was not present to reply. He felt sure that no one could have dealt more thoroughly with the subject of diarrhœa than Dr. Walker had. It must be realized that colitis was a very vague term which could not always be placed in the same category as other words ending in "itis". This was particularly true in regard to mucous colitis. He had found that the patient was much happier if he was told that he was suffering from colitis rather than diarrhœa. He would go away feeling at any rate that the medical practitioner knew his job. Dr. Blackburn usually tried to tell these patients that they did not have colitis.

Dr. Blackburn mentioned the importance that was often attached to the presence of mucus in the stool. A quantity of mucus was secreted from the upper respiratory passages

when a person was suffering from a cold, but this did not cause any great worry. Further, the third or fourth motion passed by a child who had been given a dose of castor oil was sure to contain mucus. When there was increased activity of the colon there was increased secretion of mucus. Dr. Blackburn said that he was in the habit of telling patients not to look behind. Whatever the cause of his condition, nothing made the patient more nervous than regular contemplation of a stool containing mucus. If a nervous patient saw mucus in his morning stool, he was unhappy for the rest of the day. If no mucus was observed, his mood tended to be happier. Dr. Blackburn mentioned the type of colitis characterized by an early morning attack of diarrhoea and perhaps no bowel action for the remainder of the day, save occasionally after meals. Patients affected with this type of diarrhoea should be encouraged to make a great effort voluntarily to control the action of their bowels. He had found that if they were instructed to endeavour, after having a free morning bowel action, to restrain themselves and fight against the urge to have further actions during the remainder of the day, this helped in their recovery. At the same time he usually administered a mixture containing bromides and belladonna. Patients suffering from mucous colitis should be allowed to eat anything they wished to eat. Some people became obsessed with the necessity for dietary restrictions. He had seen a patient who had come from England where she had been treated by a Harley Street physician who had provided her with a long list of things that she should not eat, and a much shorter list of things that she could eat. The patient had become emaciated. Dr. Blackburn had told her she would probably be better if she were to eat all the things on the list of prohibited articles and not to eat those which had been mentioned as permissible. As a result she had resumed her social life, having meals with her friends and eating what came her way; she had become plump and her colitis had not returned.

Dr. Blackburn remarked that it was not at all surprising to see cases of amebic dysentery in New South Wales. The wonder was that it was not more common. Amebic dysentery was endemic in all the States of the Commonwealth. In the treatment of this disease one method had not been mentioned, which, he believed, was very useful in resistant cases. This was a South American treatment and consisted in the administration of bismuth subnitrate in a dose of three drachms five times a day. He remembered one patient from the Pacific Islands who had recovered after the administration of emetine and emetine bismuth iodide. The patient had then suffered a relapse and had become very weak and ill. The administration of more emetine had been followed by acute emetine neuritis. Dr. Blackburn had then administered bismuth subnitrate, and within two weeks the patient's stools had become normal; two years later there had been no relapse. He had seen similar satisfactory results in the treatment of ulcerative colitis. Dr. Blackburn was surprised that some of the speakers had appeared to be quite cheerful when discussing the subject of ulcerative colitis. To his mind this was a very serious disease and often caused death. It must be remembered that there were different kinds of ulcerative colitis. The acute type was very similar to acute bacillary dysentery. Others could be described as ulcerative colitis only because blood was passed in the stools. In these latter cases, if treatment was applied to the lower part of the bowel, recovery was the rule. He had seen a case of acute ulcerative colitis not long before. The patient was a woman who had been successfully treated for an acute attack by Manson-Bahr in London. On her return to Australia she had become very ill and had passed great quantities of blood. The administration of "Yatren" and bowel lavage had caused no abatement of the symptoms. It had been impracticable to employ a great deal of bowel lavage owing to the profuse bleeding. The question of surgery had been debated, but large doses of bismuth had been tried with remarkable effect. Eighteen months later the patient was still quite well. Another useful method of treatment was the administration of bismuth by the rectum. Half an ounce of bismuth car-

bonate was suspended in four fluid ounces of water and was run in through a funnel and tube.

Most of the speakers had referred to mucous colitis as though it were associated with diarrhoea. In many cases it was associated with constipation. Motions were often very frequent, but usually were small and hard.

A MEETING OF THE VICTORIAN BRANCH OF THE BRITISH MEDICAL ASSOCIATION was held at the Geelong Hospital, Geelong, Victoria, on March 19, 1932. The meeting took the form of a series of clinical demonstrations by members of the honorary staff. The first part of this report was published in the issue of July 23, 1932.

Pseudo-Hypertrophic Muscular Paralysis.

DR. C. W. J. RAIT showed a male child, aged nine years, who had been seen first two years previously, when he complained of progressive weakness in his lower limbs. He was a breast-fed child, weaned at the age of seven months, then fed on "Glaxo" for three weeks, then on cow's milk. He was given plenty of malt and cod liver oil; he cut his first tooth at the age of twelve months, and walked at fourteen months. During the early stages of walking he overbalanced a good deal, tired quickly when out walking and never attempted to run. At five years he started school. It was then noticed that he had difficulty in climbing the slightest step and he appeared short of breath on the least exertion. During the first few months at school he complained that the muscles of his legs became "knotted" and painful. He became thinner about this time and it was noticed that his shoulder blades were prominent, and his back arched. His calves were very large, and his right foot tended to turn inwards. He could not now climb stairs without putting his hand on his knee to help himself; even then he could only climb two or three steps. If at any time he happened to fall he found it very difficult to rise.

The attacks of cramp in his legs became more frequent and he referred the pain to his right hip joint. This was examined by X rays, but no abnormality was detected.

The only other illnesses he had had were measles at the age of twelve months and adenoids (removed surgically) at five and a half years. There was nothing of importance in the family history.

The child stood with his legs apart and his shoulders thrown back, giving a pronounced kyphosis. The *latissimus dorsi* and pectoral muscles were atrophied, while the *infra-spinati* and muscles of the calves were hypertrophied. He had the typical winged scapulae, and in rising from the supine position he first turned over to his abdomen and then "climbed up himself".

The diagnosis was pseudo-hypertrophic paralysis. No treatment was of any effect. Liver extract and arsenic were administered and massage employed.

Comments were requested as to diagnosis, prognosis and treatment.

DR. R. P. McMEEKIN stated that the case was certainly one of pseudo-hypertrophic muscular dystrophy. It was the third he had seen without a family history. Though usually transmitted through the mother to males, transmission through the father apparently had occurred in one of his cases, and this was supported by the experience of Dr. Gordon Holmes, to whom he had written. Dr. Holmes had advised that the patient should not be allowed to marry. The duration of the disease, however, was usually short—a few years; it occurred always in young children.

DR. H. D. STEPHENS agreed with the diagnosis, which could be made on the history alone. In this case it appeared possible that the father had suffered from a peroneal dystrophy; this was of interest in the light of Dr. McMeekin's statement as to inheritance through males. Regarding treatment, he could make no suggestion. Eight years previously wealthy parents had sought advice all

over the world for their child, who was suffering from this disease; but in spite of all treatment, the paralysis had reached the intercostal muscles, and bronchopneumonia would eventually terminate the condition. Some patients had lived till over thirty years of age. Endocrine treatment had been tried, but it was ineffective.

Osteitis Deformans.

DR. R. N. SCOTT GOOD showed a male patient, aged sixty-four years, whose leg had been crushed between trucks fifteen years previously. He had been treated as an out-patient for one month and had then returned to work at the salt works. In the previous two years he had complained of some difficulty in weight bearing, with an increasing amount of anterior and external bowing of the left tibia. There was some very slight "toothache" pain at times. X ray examination revealed thickening of the periosteum of the tibia, and "honeycombing" of practically the whole of the shaft, possibly the result of chronic osteomyelitis. The patient had had no previous illness.

The patient was short in stature. There was marked anterior and lateral bowing of the left leg. There was some undue prominence of both clavicles. There was no involvement of the vertebrae nor any increase in the size of the head. The systolic blood pressure was 185 and the diastolic 105 millimetres of mercury. There was some thickening of the radial arteries. No abnormality was detected in the nervous system.

DR. VICTOR HURLEY said the case was of academic interest to radiologists. Was it *osteitis fibrosa* or *osteitis deformans*? It had been recently found that in cases of *osteitis fibrosa*, particularly in the generalized variety, tumour or lesion of the parathyroid glands was present. In his opinion, this case rather resembled Paget's disease. He could feel no abnormality in the neck. He referred to the work of Hunter on calcium deficiency in connexion with *osteitis fibrosa*, and to a foreign case in which Hunter had insisted, from a study of the published report, that a parathyroid tumour was present, although none could be palpated. The subsequent operation had confirmed his diagnosis and resulted in the patient's recovery.

DR. KEITH HALLAM referred to Dr. Hurley's remarks, and said he had worked with Hunter and Walton with their parathyroid cases, of which they had collected six to date. Dramatic results had followed the operations. He recalled the case of a London policeman, brought to the London Hospital, in whom Hunter had demonstrated the presence of hypercalcaemia and hyperphosphatemia. A parathyroid tumour had been removed from the neck, low down, almost under the clavicle. In three or four months the bones had become normal and complete recovery had resulted. He emphasized the urgency of differentiating Paget's from fibrocystic disease, in which the cysts were seen to be limited in the X ray pictures. This case, he considered, was one of localized Paget's disease. When doubt existed, the urinary and blood calcium content should be investigated.

DR. A. E. COATES considered the case was one of *osteitis deformans*. Paget's original patient had died of sarcoma, and, as students, they had been warned to watch for sarcoma in Paget's disease. He had seen such a case recently.

DR. M. D. SILBERBERG had recently seen a case of spontaneous fracture in *osteitis deformans*. Blackburn, of Sydney, had noticed improvement following the administration of vitamin D in the form of "Radiostoleum", which increased calcium retention. In later life the disease had been frequently associated with arteriosclerosis, as in this case, and he wondered whether there was any connexion between the two conditions.

DR. D. W. CHAPLIN mentioned that he knew one patient in London who had survived for seventeen years; the skull had been chiefly affected, in addition to both legs, radii and clavicles. Each year for ten years the patient had required a hat one size larger than the previous one. Dr. Chaplin considered spontaneous fracture more frequent in fibrocystic disease, which was found more particularly in the neighbourhood of joints.

Correspondence.

PARALYSIS OF THE LOWER EYELID DUE TO LEPROSY.

SIR: When at Honolulu the surgeons attending the Pan-Pacific Surgical Conference visited the Kalihi Receiving Station for Lepers close to Honolulu, and incidentally received much information.

The ophthalmologists present were interested to know that one of the comparatively early appearances in leprosy was paralysis of the lower eyelid. As none of us had seen an isolated paralysis of the lower eyelid, the question naturally arose respecting its causation.

Recently I wrote to Dr. Pinkerton, who is the Honorary Secretary of the Pan-Pacific Surgical Conference and is the Ophthalmologist for the Leper Station, and asked him what explanation of such an unusual condition was feasible. I have just received his reply, which I quote hereunder:

In my investigation of leprosy lesions of the eye, extending over twelve years and involving more than eight hundred cases, I have concluded that, in lesions involving the facial nerve, we frequently observe defect in the upward movement of the lower lid long before the whole lid is affected. I believe from my clinical observations that the facial nerve may be involved either as a whole or in its various terminal ramifications. I believe from the same observations that the levator is not involved in most cases, but on the contrary it is the orbicularis alone or chiefly that is affected, and this condition may exist alone or in connexion with a more extensive involvement of the nerve as a whole.

I have never seen a case where I considered the eye muscles involved as a result of leprosy, the intrinsic muscles only sharing in a process involving the ciliary body, which is a common complication. The extrinsic muscles are not involved, except in those cases of unusual acute oedema of the face and orbit, indirectly.

The circumstances raise anew an explanation of the fact that the lower eyelid moves upward when the eye is rotated upward.

In the eighties Messrs. Lang and Fitzgerald gave the explanation which has so far held good. It was in effect that the upper lid pulled the lower lid upward by attachments at the margin of the two lids. Possibly fixation by the orbicularis plays a part. But the issue cannot be regarded as finally settled.

Yours, etc.,

J. W. BARRETT.

103-105, Collins Street,
Melbourne,
May 23, 1932.

ANTHRAX.

SIR: With reference to my report of a case of anthrax published in THE MEDICAL JOURNAL OF AUSTRALIA of June 18, I made the statement that "anthrax is a rare disease in man in Australia at the present time" and that "no Australian case has been reported since Cleland's in 1923".

Unfortunately I was under a misapprehension as to the frequency of the occurrence of the disease. Though quite aware that cases had occurred in Australia since 1923, I did not know that there had been a very full publication of cases in the "Sixteenth Report of the Microbiological Laboratory (Government Bureau of Microbiology)" for the year 1925, from the "Report of the Director-General of Public Health of New South Wales", 1925. It appears that a large number of cases occur in New South Wales, both from shaving brushes and from anthrax infected hides, and I am much indebted to the Principal Microbiologist of New South Wales for forward-

ing me a copy of the above report, in which there is an excellent series of photographs of the types of brush found infected.

Yours, etc.,

IAN HAMILTON.

Adelaide Hospital,
Adelaide,
July 4, 1932.

PYELOGRAPHY.

SIR: Let me add a line to the excellent paper of Dr. Maitland. I had a patient with a tumour of the cortex of the left suprarenal body. The tumour could not be palpated, but when Dr. Oxenham did an intravenous pyelogram, the right kidney showed nothing, but the left kidney was seen to be displaced a little downwards and outwards. This told me which side to attack. The Americans call this ptosis of the kidney, and while there was nothing the matter with the kidney, its slight displacement was sufficient to guide one to the left side.

Yours, etc.,

W. J. STEWART McKAY.

227, Macquarie Street,
Sydney,
July 4, 1932.

DIATHERMY AND GENERAL PARALYSIS.

SIR: I have been greatly interested in the excellent account of the diathermy treatment of general paralysis of the insane by Dr. Guy Prior, and I should like to congratulate him upon the thoroughness of his observations.

Having had very little experience of the treatment of general paralysis of the insane by the malarial method, I cannot attempt to discriminate between the latter therapy and that of diathermy, but it would seem to me that no small credit for the patient's improvement should be allowed to the physical action of the diathermic pyrexia.

On this matter I may perhaps speak with some confidence, since I have been using a pyrexial method for the treatment of acute and chronic rheumatic conditions, as well as the milder manic-depressive states, melancholias, and some psychoneurotic disturbances, for a number of years.

Originally I employed diathermy, but, as the results were not always as gratifying as might be expected, I adopted the Wilde pyretic couch.

This appliance, designed and used in various forms in the Lansdowne Hospital, Bath, by the late Percy Wilde, is manufactured by the Cox-Cavendish Electrical Company, 105, Great Portland Street, London. It may well be said to be the basic treatment *par excellence* for all rheumatic and gouty conditions, and has now achieved popularity in many countries.

In principle it differs from the usual steam or vapour bath in that a moist, warm air, of a humidity of approximately 80 and a temperature of not more than 106° F., is used. The patient lies on a thick coir mat, which in turn is covered with a blanket and sheet. These, intervening between the patient and the steam condensers, serve as filters and permit moist, warm air, at a temperature which can be regulated, to fill the couch.

Treatment is given daily for about forty minutes, during which time the patient's temperature may be raised to 103° or 104° F., and brisk action of the skin always follows. A tepid or cold shower or sponge-down is then administered, and the patient rests for ten minutes before dressing.

It is claimed that the sweating thus produced: (i) approximates most closely to that of natural exercise, (ii) is tonic and refreshing, (iii) causes no nervous exhaustion, and (iv) has a heavier acid and salt content than that effected by any other artificial means. The pyrexia undoubtedly is a powerful adjuvant towards restoring normal metabolic functioning.

However, my purpose is not to extol the use of the pyretic couch in rheumatic diseases, but to suggest that such an appliance, which is eminently suited for institutional work, might be found more valuable in the treatment of general paralysis of the insane than diathermy.

Sundell, using the couch in the Prince of Wales Hospital, London, and the "Dreadnought", Greenwich, for treatment of rheumatism and arthritis, maintains a body temperature of 104° to 106° F. for two to three hours daily without any ill-effects, and his therapeutic results are excellent.

Wilde gave over a million treatments without any untoward happening.

I do not know whether or not the pyretic method has ever been used in general paralysis of the insane, but it would seem to have the advantages of, first, being a reliable and pleasant means of inducing pyrexia, secondly, being quite free from accident or danger, and thirdly, being cheaper in initial cost than a powerful diathermy machine.

Yours, etc.,

E. HASLETT FRAZER.

231, Macquarie Street,
Sydney,
July 4, 1932.

AMENORRHOEA AND HORMONE THERAPY.

SIR: In reference to case report submitted by Dr. Darby Thomas, of Melbourne, "A Case of Amenorrhœa with Response to Hormone Therapy", published in THE MEDICAL JOURNAL OF AUSTRALIA, June 25, I would like to draw attention to the fact that at the present stage of our scientific knowledge there only exists one ovarian hormone, generally called "œstrin", which is different from the *corpus luteum* hormone.

The ovarian hormone is contained in "Sistomensin" only. The active substance contained in "Agomensin", on the other hand, has not been identified. We know that it possesses pharmacological and therapeutic properties different from those of the ovarian hormone; but as the active substance contained in "Agomensin" has not been determined, it is not possible to state definitely that it is a hormone.

For this reason it would have been more correct to have modified the title as follows: "A Case of Amenorrhœa with Response to Ovarian Therapy", and the last sentence as follows: "The response to treatment was so marked on each occasion that it must be attributed to an active ovarian substance present in 'Agomensin'."

With the present title of the paper the reader draws immediately the conclusion that it refers to the ovarian hormone, that is, "œstrin", which is not the case.

Yours, etc.,

J. M. HARKNESS,

The Society of Chemical Industry
in Basle. "Ciba."

Sydney,
July 6, 1932.

DIATHERMY OF TONSILS.

SIR: The article appearing in the issue of July 2, 1932, by Dr. Graham Brown and Dr. Duhig will surely do much to place the treatment of diseased tonsils by diathermy in its proper place. Generally speaking, it should only be used where there are definite contraindications against the use of a local or general anæsthetic.

I think that Dr. Brown has clearly demonstrated the first five points he set out to do in his paper, and I am in complete accord with him on every point. His experience, I take it, is mainly on cases treated in Brisbane, and it agrees with my observation on cases treated in Melbourne.

I am convinced that pain is not a small factor in such treatment. That tonsillar tissue is left behind in the majority of cases, with subsequent trouble, surely admits of no argument.

The length of time required for the destruction of the tonsil converts many patients into neurotic wrecks. I have known patients admit that they have had sixteen applications of diathermy and yet they have septic tonsillar remains. Personally I should feel rather ashamed of my efforts in such cases.

I am sure that all who read and digest Dr. Brown's excellent paper will be amply rewarded.

Yours, etc.,

JAMES M. BAXTER, M.D.,
Surgeon to Ear, Nose, Throat
Department, Saint Vincent's
Hospital, Melbourne.

July 7, 1932.

NEW SOUTH WALES METROPOLITAN HOSPITALS CONTRIBUTION FUND.

SIR: In the *Sunday Sun* and *Guardian* of June 19, 1932, wide publicity is given to the Metropolitan Hospitals Contribution Fund. Citizens are invited to contribute sixpence per week, in return for which they are promised any one of ten benefits. Benefit number 1 is: public ward patients get service, treatment, accommodation, everything free. It is asserted that "everybody realizes that public hospitals are not charitable institutions". In THE MEDICAL JOURNAL OF AUSTRALIA of November 28, 1931, the British Medical Association, New South Wales Branch, endorses cooperation in this scheme, provided the interests of the medical profession were safeguarded. The two large cities, Sydney and Melbourne, form the reservoir from which doctors for all parts of the Commonwealth are drawn. The economic condition of practice throughout the Commonwealth therefore depends very largely upon the conditions prevailing in Sydney and Melbourne. The approval of this scheme by the New South Wales Branch of the British Medical Association therefore vitally interests the medical profession throughout Australia, and there are many members extremely anxious to know what are the safeguards which would protect the interests of the profession when endorsement has been given to a scheme which entitles contributors and their dependants to service in public utilities maintained for them by their contribution of sixpence per week.

Yours, etc.,

D. M. EMBELTON.

37, Collins Street,
Melbourne,
July 7, 1932.

"AVERTIN."

SIR: It is probably not unfitting that yet another "Devil's Advocate" should be heard in the process of the canonization of "Avertin" in the calendar of great recent advances. It does seem that the repeated recitation of the happy and glorious experiences of its enthusiastic advocates is apt to hypnotize the rest of the world into believing that this drug is in fact a very wonderful innovation. As the result of some experience, I am now wondering if it is even worth the extra trouble of its administration. There are many unpleasant experiences that most people who use "Avertin" have at some time to endure. First, it would appear that the acid test of the skin incision is the only really reliable method of estimating the depth of anaesthetization. It so often happens that this rouses the pallid form of the patient to the most disquieting gesticulations, which unceremoniously precipitate all instruments on to the floor. It so often happens that the anaesthetist is apt on these occasions to feel a little responsibility for the shortcomings of "Avertin" and to compensate by the administration of too little ether,

what time the surgeon does his part in smoothing things over by struggling with incomplete relaxation.

Nor have the after-effects been happy. The time of recovery to normal is very considerably longer and often more distressing than the usual recovery from inhalation anaesthesia.

The fact that the patient has amnesia for all this is indeed happy, but I feel that until something is done to induce amnesia in the surgeon and his assistants also, many of us will use less cumbrous forms of basal hypnosis. For if we are to regard it as merely a preanaesthetic, surely there are many others easier of administration and safer to use. Possibly it is the method of administration that appeals to people's sense of the wonderful.

The more one sees of such performances as I have described, the more one values the trite saying of a very wise and experienced surgeon: "If you have to use poisons, use one at a time, and use the best."

Yours, etc.,

DOUGLAS MILLER, F.R.C.S.

185, Macquarie Street,
Sydney,
July 7, 1932.

SIR: I had hoped to keep out of this "Avertin" controversy, but the whole question is not one merely of "Avertin", it really embraces the error of using some of the basal anaesthetics, such as "Avertin", "Luminal", "Amytal" et cetera, as anaesthetics *per se*. In other words, using any form of general anaesthesia in which the anaesthetist loses control. What I have said about "Avertin" applies with equal force, if not more so, to the unnecessary use of spinal, intravenous or rectal forms of anaesthesia.

There is not one operation on the human body, from the crown of the head to the sole of the foot, that cannot be performed with advantage in a stage of anaesthetic sleep, provided there has been correct premedication and provided the control anaesthesia is some form of inhalation anaesthesia.

Premedication should never under any circumstances be such that the patient is brought on to the operating table in a stage of "down and out", but it should be such a stage in which the patient has the feeling of "Oh, just let me drift off to sleep, Doc., that's all I want". "I am quite happy, I just want to go to sleep and am not worrying about anything." The simple anaesthetics and the simple methods of administering those anaesthetics are the correct and best methods, in spite of the present outcry for machines. Stopcocks, valves and percentages never have and never will make an anaesthetist—there is far more than that required.

There are far too many anaesthetic deaths today, either direct or indirect. They are not always the fault of the anaesthetist; in many cases the surgeon is a contributory factor. He either does not know or appears not to care how things are, as long as he gets what he considers an ideal method. I do not contend and never have contended that under the various forms of uncontrollable anaesthesia, such as intraspinal, intravenous, rectal or the basal anaesthetics, you do not and cannot get a perfect condition from the operator's point of view for doing the job. That, however, is not the point. The point is that the surgeon or anaesthetist, and it makes no difference how eminent a surgeon or anaesthetist he may be, who employs some form of general anaesthesia in which control is lost, is not playing cricket by his patient; he is worse than a charlatan, because he ought to know better.

The duty of the anaesthetist is not primary towards the surgeon; his duty is towards the patient, first and always. It is the duty of the anaesthetist from the time he sees his patient prior to operation, right throughout that operation and until he sees him back into bed and satisfies himself that all's well, to take control of that patient as far as his life is concerned.

That patient's life is mine, not the surgeon's, and I surrender that right to no one. It is for the anaesthetist not only to handle that patient correctly during induction, but to maintain during operation a stage of anaesthesia

that cuts off shock, or to use such preliminary medication that such shock is reduced to an absolute minimum.

It is for him to handle the patient throughout the operation, not in a stage of so-called uniform anaesthesia, but in a stage in which he uses the absolute minimum of anaesthetic combined with a perfect surgical anaesthesia, and all this under control. To do this the anaesthetist must not only know his drugs, but he must also know his anatomy and his nerve areas and roughly the various stages of the operation that the surgeon is doing or attempting to do.

All this can be done if men will only learn their jobs and learn the handling of the simple methods correctly. You can get exactly the same advantages with the correct methods of handling the major anaesthetics, chloroform and ether, and provided there has been moderate premedication, as you can under the so much vaunted gaseous anaesthetics. In fact better, because you get better relaxation. In other words, we have been able today to cut down our consumption of the major anaesthetics and at the same time give perfect relaxation, so that post-operative vomiting, bronchopneumonia *et cetera* are horrors of the past.

One reason why we are forced so much today into the freak and mechanical methods of anaesthesia is the fact that anaesthetic work, like medicine, is becoming commercialized. Many medical men cannot write a decent prescription; they do not even know the compatibility or otherwise of various drugs and have to resort to some proprietary formula brought to their notice by an over-enthusiastic salesman. And the same with anaesthesia.

Mechanization of our anaesthetic methods will not lower our anaesthetic fatalities. If we would only learn the decent handling of simple anaesthetics by simple methods, neither our surgeons, ourselves nor our patients would be the losers, though we might not be so impressive or look so formidable.

Yours, etc.,

R. W. HORNABROOK.

190, Collins Street,
Melbourne,
July 9, 1932.

PROSTATECTOMY WITH CLOSURE OF THE BLADDER.

SIR: Some criticism is called for in regard to Dr. Close's article entitled "Prostatectomy with Closure of the Bladder, with Reference to a Modification of the Harris Operation", appearing in THE MEDICAL JOURNAL OF AUSTRALIA on July 9, 1932.

The operation which Dr. Close describes is an entirely different one from that which bears my name, and can scarcely be described as a modification of it. There are two essential points in my operation which I should like to stress, namely, the complete control of hæmorrhage by suture and the reformation of the prostatic urethra. The operation was devised with these two main objects in view. Neither of these two objects is either sought or attained in the operation described by Dr. Close.

The trigonal "tongue" which is sewn down to the floor of the prostatic cavity is a strong and muscular body, and not merely a mucosal flap, as Dr. Close states in his paper. This step has provided in my hands an absolute safeguard against "ledge formation", whilst its omission has more than once been followed by this complication.

With regard to Dr. Close's observation that with my technique he has sometimes been unable to control oozing, my earlier experience was that this happening was invariably due to inaccurate placing of, or insufficiency in number of, the postero-lateral hæmostatic sutures (which control the gross bleeding), or an insufficiently wide bite of the anterior obliterative sutures, with the result that a large enough body of muscular tissue was not inverted into the prostatic cavity to obliterate the cavity and thus control oozing. Later experience has corrected these earlier defects. The minor alterations in technique which were rendered necessary, and which I have now employed for nearly three years, will appear in print at an early date.

They include, in essence, the placing of a rather greater number of hæmostatic sutures than were originally described, and of the taking of as wide a bite of tissue in the anterior sutures as it is possible for the needle to hold. The result is not only a better filling in of the prostatic cavity, but also a wide inversion or rolling in of the roof of the prostatic cavity, thus largely reforming the side walls of the new prostatic urethra. At the termination of the operation, as now carried out, there is no visible raw surface and no bleeding.

I have always been extremely careful that all bleeding is controlled before the catheter is passed. The catheter itself plays no part in the control of hæmorrhage, ample room always being left around it to allow of drainage upwards into the bladder from the remnant of the prostatic cavity. Experience has proved that tight suturing of the prostatic rim round a rubber catheter predisposes to sloughing of the contiguous parts and to secondary hæmorrhage. One would expect the elastic traction provided by Dr. Close to increase this liability. Post-operative ledge formation will also, I believe, be a source of worry with Dr. Close's technique.

There have, as with all new procedures, been several suggested modifications of the original, but as far as I am able to judge, none so nearly reproduces the normal anatomical relationship as the original does, and practically all result in the formation of some type of iris diaphragm fitting tight round the catheter; and none seems to me likely to be so free from annoying post-operative sequelæ as the original has proved to be.

I trust that, having gone so far, Dr. Close will not abandon the original technique on account of some preliminary disappointments (the cause of which he will readily overcome if he so desires) in favour of an operation which may at the moment seem to him easier of application, but which will, I believe, in the long run lead to disappointment.

If Dr. Close will honour me with a visit when he is next in Sydney, I shall be very pleased to resume this discussion with him.

Yours, etc.,

S. HARRY HARRIS,
M.D., Ch.M., F.R.A.C.S.

185, Macquarie Street,
Sydney,
July 15, 1932.

DRAINAGE WITH IRRIGATION AFTER PROSTATECTOMY.

SIR: I have to thank Dr. Morton for sending me his courteous reply to my criticism.

It is very pleasing to know that he had no intention of advocating the cautery as a substitute for ligature or suture to control hæmorrhage. The method of passing the perineal drainage tube described in his original paper (namely, "... make a small mid-line incision in the perineum behind the bulb. Shove a closed clamp through into the prostatic pouch, guided by two fingers of the left hand passed into the bladder from above") has a very different sound from that which he describes in his letter. The risk of injury to the external sphincter, however, will, in my opinion, still remain a very real one.

It is, of course, a *sine qua non* that each surgeon is entitled to use the technique which gives him the best results, and the fact that a surgeon of Dr. Morton's experience has found the method of operation which he advocates to yield, in his hands, the best results of any he has employed, is ample warrant for his employment of this technique.

I would repeat, however, what I wrote in my previous letter, that any method of prostatectomy will yield a certain proportion of successful results. After an experience of now nearly a thousand prostatectomies, I am firmly convinced that no method which fails to control hæmorrhage will, in a large series of cases, yield results either as to mortality or morbidity in any way comparable to those obtainable by methods which do control hæmorrhage.

I would prefer not to cloud the issue by the introduction of extraneous matter, but, as Dr. Morton has in his letter introduced the subject of the fibrotic prostate in order to advocate his method of combined perineal and suprapubic prostatectomy, I shall conclude by stating that urologists generally do not find it either necessary or advisable to perform a prostatectomy for this condition, a prostatic punch operation through the urethra, with or without diathermy, amply fulfilling indications.

Yours, etc.,

S. HARRY HARRIS,

M.D., Ch.M., F.R.A.C.S.

185, Macquarie Street,
Sydney,
July 15, 1932.

Obituary.

HENRY TRUMAN KELSALL.

WE regret to announce the death of Henry Truman Kelsall, which occurred at Buckland Hill, Western Australia, on May 19, 1932.

Dr. F. A. Hadley writes:

Few of Dr. Kelsall's early colleagues in Western Australia now remain with us, otherwise one of them would probably have been able to speak with greater authority than I can about his earlier activities.

However, many years have rolled on since my own first arrival in this State and from the day of my landing I have enjoyed his most intimate friendship. I found a man with tireless energy, with many and varied interests, and a man who felt that every hour which could be spared from work should be filled with active exercise. He played both cricket and tennis well and indeed until a few years ago even young men found it hard to snatch a set from him. He had the art of spreading his enthusiasm to others, to the great benefit of games as well as to his professional brethren in more serious matters.

Dr. Kelsall was educated at the United Service College, Westward Ho, England, and at the London Hospital, taking his M.D. degree at London University in 1888. Then, after being resident at "Moorfields", he joined the Navy for a short period, but resigned to take up work with the North Sea fishing fleet, being influenced to do so by his lifelong friend, Sir H. Grenville, of Labrador fame. In those days, even more than now, life was very strenuous amongst those storm-tossed trawlers and the successful treatment of the many severe accidents very difficult. He carried the broad vision of the sailor on to his work on land.

He came to Western Australia in 1900 and soon found himself with a big practice. Always fond of eye work, towards the end of his medical career he devoted himself completely to it.

Dr. Kelsall was one of the Foundation Members of the Western Australian Branch of the British Medical Association and was one of the first presidents. He also for many years was a member of the board of the Perth Hospital, and was on the honorary staff, first as surgeon and later as ophthalmic surgeon. He was a member of the State Medical Board from its inception, and for many years had held the position of chairman and acted in that capacity at a meeting only a few weeks before his death. There are many other institutions which owe him a deep debt of gratitude.

He married Miss Leake, the daughter of George Walpole Leake, Q.C. He had a large circle of friends, especially among the older families of the State. In latter years he retired and lived on his farm near Moora until, with failing health, he moved to within sound of the sea at Cottesloe.

HERBERT LILLIES.

HERBERT LILLIES, who died on May 30, 1932, was the son of Dr. George Lillies, of Chudleigh, Devon, England. He

was born at Chudleigh on December 17, 1857. He received his schooling at All Hallows School, Honiton, then proceeded to Saint Bartholomew's Hospital. While a student he had the privilege of attending Lister's first address on antiseptics. After obtaining the diplomas of M.R.C.S. (London), L.R.C.P. (Edinburgh), he was appointed house surgeon at the Devon and Exeter Hospital. After a term of residency he commenced general practice as assistant to his father at Chudleigh. He came to Australia in 1885. After acting as *locum tenens* for Dr. Rankin, of St. Kilda, Melbourne, he commenced practice at Armadale, where he remained until the time of his death. During his later years he was assisted in his practice by his son, Dr. G. L. Lillies. His passing will be mourned by a large circle of friends.

Dr. R. H. Morrison writes:

It was in the early hours of a winter's morning in my first month of private practice that I first met Dr. Herbert Lillies. He had been called to my assistance in a case of eclampsia, and his support was greatly appreciated. From then until his death we remained firm friends. He was a man of Devon, tall, fresh complexioned, and with the kindest of expressions. He had a marked limp resulting from a fractured thigh. He had come from London in 1886 and settled in Armadale, where he rapidly acquired a very extensive general practice in the best residential suburbs of Melbourne. He was more especially favoured as an obstetrician and as the trusted family physician. Surgery never appealed to him. For many years he was physician to out-patients at the Alfred Hospital until, threatened with lung trouble in 1899, he acted on the advice of his friends and resigned in order to devote all his time to his private practice. The calls on his services were even then more exacting than he could fulfil, and he found it necessary to take extended rest for twelve months, most of which was spent in England. On his return he attempted to carry on at the same high pressure as formerly, but his physique was not equal to the strain, and he had gradually, through the ensuing years, to relinquish much of his work. In later years he took into partnership his elder son and so was able to enjoy more leisure.

Lillies was truly the beloved family physician and friend, a most lovable personality, the soul of honour. I would particularly stress one of his virtues, a virtue often lacking in members of our profession: he recognized his limitations and never hesitated to suggest and seek another opinion if he thought it would be of benefit to the patient. It is safe to state that the British Medical Association never had a member more punctilious in acting up to the highest standard of professional ethics. He was a member of the Victorian Racing Club and the Victorian Associated Trotting Club and a regular attendant at their meetings. His indoor recreations were reading and bridge.

The large attendance at his funeral, of doctors, patients and friends, was evidence of a popularity which was as general as it was deserved.

University Intelligence.

THE UNIVERSITY OF SYDNEY.

A MEETING of the Senate of the University of Sydney was held on July 4, 1932.

The degree of Master of Surgery (Ch.M.) was conferred *in absentia* on Harold Baynes Cribb, M.B.

The following diplomas were awarded:

Diploma in Tropical Medicine: Gerald Douglas Broome, M.B., B.S. (Melbourne); Ian Thomas Dickson, M.B., Ch.M. (Sydney); Talbot Lewis Dunn, M.B. (Adelaide).

The following appointments were approved: Dr. A. J. Arnott as Honorary Tutor in Dental Surgery; Dr. C. H. Kellaway (Melbourne), Professor C. G. Lambie and Professor H. D. Wright as Examiners for the thesis submitted by Mr. E. L. Morgan, M.B., Ch.M., for the degree of Doctor of Medicine.

Books Received.

- HUMAN STERILIZATION: THE HISTORY OF THE SEXUAL STERILIZATION MOVEMENT**, by J. H. Landman, Ph.D., J.D., J.S.D.; 1932. New York: The Macmillan Company. Demy 8vo., pp. 359.
- THE MEDICAL ANNUAL: A YEAR BOOK OF TREATMENT AND PRACTITIONER'S INDEX**, Edited by C. F. Coombs, M.D., F.R.C.P., and A. R. Short, M.D., B.S., B.Sc., F.R.C.S.; Fiftieth Year; 1932. Bristol: John Wright and Sons, Limited. Demy 8vo., pp. 674, with illustrations. Price: 20s. net.
- BAILLIÈRE'S SYNTHETIC ANATOMY**, Parts I to XII, by J. E. Cheesman; 1932. London: Baillière, Tindall and Cox. Crown 4to., with 156 plates. Price: 42s. net.
- HANDBOOK OF BACTERIOLOGY FOR STUDENTS AND PRACTITIONERS OF MEDICINE**, by J. W. Bigger, M.D., Sc.D.; Third Edition; 1932. London: Baillière, Tindall and Cox. Demy 8vo., pp. 475, with illustrations. Price: 12s. 6d. net.
- FILTERABLE VIRUS DISEASES IN MAN**, by J. Fine, M.D., B.Sc., D.P.H., D.T.M.; 1932. Edinburgh: E. and S. Livingstone. Crown 8vo., pp. 144. Price: 6s. net.
- OLD BELIEFS AND NEW KNOWLEDGE: THE BASIS AND DEVELOPMENT OF RELIGION**, by W. Russell, M.D., LL.D.; 1932. London: John Bale, Sons and Danielsson, Limited. Crown 8vo., pp. 284. Price: 5s. net.
- MOTHERCRAFT, ANTENATAL AND POSTNATAL**, by R. C. Jewsbury, M.A., D.M., F.R.C.P.; 1932. London: J. and A. Churchill. Foolsap 4to., pp. 188, with 11 illustrations, 13 in colour. Price: 10s. 6d. net.
- RECENT ADVANCES IN PATHOLOGY**, by G. Hadfield, M.D., F.R.C.P., and L. P. Garrod; 1932. London: J. and A. Churchill. Demy 8vo., pp. 402, with 67 illustrations. Price: 15s. net.
- POCKET MONOGRAPHS ON PRACTICAL MEDICINE: THE DISCHARGING EAR**, by A. G. Wells, B.S., M.B., D.P.H.; 1932. London: John Bale, Sons and Danielsson, Limited. Foolsap 8vo., pp. 89. Price: 5s. net.

Medical Appointments.

The undermentioned have been appointed Members of the Medical Board of South Australia: Dr. R. S. Rogers (B.M.A.), Dr. A. M. Cudmore (B.M.A.), Dr. E. A. Johnson (B.M.A.), Dr. B. H. Morris (B.M.A.), Dr. H. H. E. Russell (B.M.A.).

Dr. F. K. Mugford (B.M.A.) has been appointed Assistant Medical Officer in the Children's Welfare and Public Relief Department, to be also Visiting Medical Officer of the Yatala Labor Prison, South Australia.

Dr. K. Brocke-Cowden has been appointed a Resident Medical Officer at the Adelaide Hospital, South Australia.

Dr. S. B. Forgan (B.M.A.) has been appointed a Member of the Medical Board of Port Pirie, South Australia, under the provisions of the *Workmen's Compensation Act Amendment Act, 1927*.

Dr. C. E. Willing has been appointed District Medical Officer, Gnowangerup, Western Australia.

Medical Appointments Vacant, etc.

For announcements of medical appointments vacant, assistants, *locum tenentes*, sought, etc., see "Advertiser," page xiv.

FREMANTLE HOSPITAL, FREMANTLE, WESTERN AUSTRALIA: Resident Medical Officer.

LAUNCESTON PUBLIC HOSPITAL, TASMANIA: Resident Medical Officer (male).

ROYAL ALEXANDRA HOSPITAL FOR CHILDREN, SYDNEY, NEW SOUTH WALES: Resident Medical Officers.

THE BRISBANE AND SOUTH COAST HOSPITALS BOARD, QUEENSLAND: Honorary Clinical Assistant.

Medical Appointments: Important Notice.

MEDICAL practitioners are requested not to apply for any appointment referred to in the following table, without having first communicated with the Honorary Secretary of the Branch named in the first column, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

BRANCH.	APPOINTMENTS.
NEW SOUTH WALES: Honorary Secretary, 135, Macquarie Street, Sydney.	Australian Natives' Association. Ashfield and District United Friendly Societies' Dispensary. Balmain United Friendly Societies' Dispensary. Friendly Society Lodges at Casino. Leichhardt and Petersham United Friendly Societies' Dispensary. Manchester Unity Medical and Dispensing Institute, Oxford Street, Sydney. North Sydney Friendly Societies' Dispensary Limited. People's Prudential Assurance Company Limited. Phoenix Mutual Provident Society.
VICTORIAN: Honorary Secretary, Medical Society Hall, East Melbourne.	All Institutes or Medical Dispensaries. Australian Prudential Association, Proprietary, Limited. Mutual National Provident Club. National Provident Association. Hospital or other appointments outside Victoria.
QUEENSLAND: Honorary Secretary, B.M.A. Building, Adelaide Street, Brisbane.	Brisbane Associated Friendly Societies' Medical Institute. Mount Isa Mines. Toowoomba Associated Friendly Societies' Medical Institute. Chillagoe Hospital. Members accepting LODGE appointments and those desiring to accept appointments to any COUNTRY HOSPITAL are advised, in their own interests, to submit a copy of their agreement to the Council before signing.
SOUTH AUSTRALIAN: Secretary, 207, North Terrace, Adelaide.	All Lodge Appointments in South Australia. All Contract Practice Appointments in South Australia.
WESTERN AUSTRALIAN: Honorary Secretary, 65, Saint George's Terrace, Perth.	All Contract Practice Appointments in Western Australia.
NEW ZEALAND (Wellington Division): Honorary Secretary, Wellington.	Friendly Society Lodges, Wellington, New Zealand.

Editorial Notices.

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